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To Stanley Cobb

Who Embodies the Spirit Underlying this Journal

On October 14th and 15th, 1960, the Massachusetts General Hospital dedicated the Stanley Cobb Laboratories for Research in Psychiatry and celebrated the 25th anniversary of the psychiatric service which he had founded.

It seems appropriate for JNMD to join this celebration by publishing the tribute to him which was delivered at the annual meeting of the American Psychosomatic Society in Atlantic City on May 5, 1955, when Dr. Cobb became the President of that Society.

ANNUAL MEETING; AMERICAN PSYCHOSOMATIC SOCIETY, ATLANTIC CITY, MAY 5, 1955

Today we are introducing Dr. Stanley Cobb as President of this Society. It is a suitable occasion for deviating from the usual informality, if only because we are honoring ourselves in having him fill this office. He has just retired from the Bullard Professorship of Neuropathology at Harvard, and from twenty years as Director of the Psychiatric Service at the Massachusetts General Hospital. This has broad significance, because his appointment to this post had given formal recognition to one of the unique features of American medicine, namely the integration of dynamic psychiatry with medical science.

Moreover, Stanley Cobb represents America in an even broader sense. He represents the confluence of many streams which flow from the earliest New England traditions; and he manifests the capacity of the true bearers of that tradition to change, and to encompass new horizons. He embodies the liberal tradition of this country, and in medical science he has carried that same liberal spirit into his clinical and experimental work. He was a naturalist before he became a clinician, a clinician before he became an experimentalist—and he was an artist even before any of this, for he has sketched and painted all his life.

A few biographical highlights add depth and poignancy to the meaning of his life for others. Between the ages of 15 and 17 he was kept out of school because of the suffering due to his severe stuttering. In those two years he had the good fortune to come under the guidance of a tutor of exceptional gifts, who helped him to become a keen observer of the flora and fauna on the banks of the Neponset River and a precise recorder of his observations. He became an ornithologist, learned taxidermy and started a museum.

Stuttering marks many members of his family, as he once told me, and his concern with this aroused in him an early interest in genetics. In fact he turned everything which another man might regard as a handicap into an intellectual and spiritual asset.

In medicine a long and fruitful association with Dr. Councilman in pathology was followed by an internship under Dr. Harvey Cushing and a then brief experience in private practice, which increased his in-

terest in psychiatry and led him to the Henry Phipps Psychiatric Clinic of the Johns Hopkins Hospital to study under Dr. Adolf Meyer. His experience in the Army in World War I rekindled his interest in clinical neurology. Consequently while on a two-year trip abroad on a Rockefeller Fellowship after World War I his interest in both the psychological and organic aspects of nervous disorders became clear. In Munich he made his first explorations into psychoanalytic psychiatry with an Adlerian analyst, while at the same time he studied brain pathology under Dr. Walter Spielmeyer. On returning to Boston he worked for a time in the laboratories of Dr. Walter Cannon and of Dr. Alexander Forbes. Finally in 1921 he was appointed Bullard Professor of Neuropathology at Harvard Medical School; and in 1929 organized a new Neurological Unit for the Boston City Hospital. Finally in 1934 he came full circle when he was appointed Psychiatrist-in-Chief to the Massachusetts General Hospital. There, with a grant from the Rockefeller Foundation, he organized a psychiatric service in a general hospital which in many ways was unique at that time.

At various points during the course of this hegira Stanley Cobb exposed himself to formal psychoanalysis in his efforts to explore the light which psychoanalytic methods could throw on the interrelationships between modern neurophysiology, neuropathology, clinical neurology, autonomic neurophysiology, psychiatry and internal medicine. The unique quality of his personality made it possible for him to exercise a profoundly integrating influence on medical science.

He has exercised the influence directly on at least a hundred younger medical scientists from America and from other lands, and on countless others through his writings.

His bibliography contains well over 200 titles on a wide variety of topics. These appeared in a series of waves or clusters. First perhaps the papers on the nesting of birds, inheritance in rabbits, electromyographic studies of neuromuscular system in humans and elephants, and an array of clinical neurological studies. Then came an increasing focus on the problems of the epilepsies, cerebral circulation, cerebral metabolism and brain pathology in general. Beginning in 1936 he wrote a series of annual summaries of the year's work in neurology and psychiatry, and in 1939 an inclusive review of stuttering. In 1938 he was one of the first among American medical scientists to become aware of the importance of the work of the ethologists, as represented by Lorenz. Gradually as the years went on came an increasing emphasis on the inter-dependence of organic and psychological variables in the production and control of illness in general, with special emphasis on such problems as stuttering, the epilepsies, rheumatoid arthritis, pain, anorexia nervosa, the GI tract, and on psychosomatic disorders and psychosomatic mechanisms in general.

One would not do justice to the range of his imaginative spirit if one omitted, even from so brief a survey, any mention of a few by-products of his creative spirit. In 1908 he had described the mid-summer birds of the Catskill Mountains; but this was not published until 1920. He wrote an account of the medical fees collected between the years 1792 and 1850 by his own great-grandfather, Dr. Archelaus Green Smith; and included a marvelous cloak-and-dagger account of how the venerable doctor had stolen a cadaver, and how he had been tried and acquitted. He wrote a Masque for a dinner to Dr. Taylor in 1926, and a play for a dinner to Dr. James Ayer in December of 1935. A note on "First Opening Gordon's Package" sounds as mysterious as the package itself. He described the history of the psychiatric illnesses of Thackeray's wife. And once again in 1945 he turned his attention back to the birds he had always loved, with a comparison of the bird popula-

tion of Massachusetts in the summers of 1945 and of 1905. (His first paper on birds had been published in 1904 when he was 17.)

In 1954, on the occasion of his retirement as Chief of the Psychiatric Department of the Massachusetts General Hospital, Stanley Cobb published a review of twenty years of his productive leadership. The title of his paper was "Psychiatric Research from 1934 to 1953," and it was devoted largely to a review of the work of others. No description of the man would be complete without comment on this, as a compelling example of one of his major traits. Modesty has been the keystone of his need to master every facet of his complex chosen field. Modesty has made him a superlative teacher and a creative writer. And modesty is also the source of the humility which has marked all of his patient and painstaking experimental and clinical investigations. Finally it was his modesty which infused his analytically guided search for the knowledge of himself in depth. Indeed it is this unique combination of modesty and dignity which is the image of the man.

Lawrence S. Kubie, M.D.

MULTIPLE SCLEROSIS: ASSESSMENT OF TREATMENT WITH A MODIFIED LOW-FAT DIET

ROY L. SWANK, M.D.[1] AND R. B. BOURDILLON, M.D.[2]

The assessment of treatment of a chronic remitting disease is notoriously difficult. With multiple sclerosis the course of disease is so variable that assessment requires the observation both of large numbers of patients on treatment and of control cases for many years. The following analysis of the value of a modified low-fat diet in multiple sclerosis is based on the senior author's series of 156 cases of which 118 followed the diet closely for from five to ten and one-half years. These cases are compared with control cases from his own series as well as from those of other authors.

MATERIALS AND METHODS: EVALUATION AND DISCUSSION

The material and the methods of study have been described previously (20–22), and full details of the diet with many special recipes and advice to patients have been published separately (23). In this study attention was paid primarily to three points; first, the frequency of relapses; second, a personal determination of the neurological status and performance of each patient at fairly regular intervals throughout the test by the same observer (R.L.S.); and third, the continued observation and periodic recording of diet by the patients, checked by the same dietitian[3] at regular intervals throughout the study.

As noted previously (22) studies were initiated in 264 patients with multiple sclerosis at the Montreal Neurological Institute. One hundred and eight of these patients were seen but a few times during a period of from a few days to several months and were lost to our study. Among the reasons for defection of these patients were difficulties in keeping in contact with us because they lived so far away, and lack of interest by the patient in the dietary regimen from the beginning or after a few months trial.

Our study has therefore been limited to the remaining 156 patients who maintained prolonged contact with us. Nine of these were later eliminated from the study for the following reasons. Two were rejected because of uncertainty in the diagnosis; and two proved to be purely progressive cases with no evidence of remissions at any time. Five cases were lost to the study early, owing to distance or other inconveniencing factors. New patients were not added to the study group after April, 1954, when the observers had moved to Portland.

THE DIET

Patients starting diet in the first years of the test (1948–1951) were allowed a daily intake of 20 to 30 grams of fat, mostly milk and animal fat. Those starting in 1952–1954 were restricted to 15 grams of animal or "saturated" fat. Margarine, shortening and hydrogenated peanut butter were considered as "saturated" fats and their use discouraged. Butter, whole milk, cheese and all other foods containing butterfat were eliminated from the diet. Fifteen grams of unsaturated vegetable oil (corn oil, cottonseed oil, olive oil, soya bean oil, safflower oil) or fish oil, and five grams of cod liver oil were added to the daily food consumption. The diet usually included 50 or more grams of protein, one egg, and possibly an additional

[1] Division of Neurology, Department of Medicine, University of Oregon Medical School, Portland, Oregon. This study was supported by Public Health Grant B-1536 and by a grant from the Multiple Sclerosis Society of Portland, Oregon.

[2] Ganges, British Columbia, Canada.

[3] Except for the first six months of the study, one and the same dietitian, Aagot Grimsgaard, has controlled the diets of the patients in this study.

five to 10 grams of "invisible" fat in bread, skim milk, and other sources. About half of the patients starting on diet in 1948–1951 changed to the new diet between 1952 and 1954.

NEUROLOGICAL GRADING

In grading the neurological status of patients with multiple sclerosis the following categories were adopted in this series:

0—The patient was normal neurologically, and his daily performance was normal.

1—His physical and mental performance was normal, but abnormal neurological signs were present.

2—His performance was impaired slightly to moderately (generally these patients worked part time or were actively ambulant; a few worked full time), and abnormal neurological signs were present.

3—His performance was severely impaired, but he was still ambulant; he was usually not able to work.

4—The patient was confined to wheel chair or bed.

5—The patient was deceased.

Results published by other authors have been compared with those of Swank by using the equivalents shown in Table 1. While only roughly approximate, the average grades thus obtained agree in their general course sufficiently to act as a useful control (shown in Figure 7). The scale of McAlpine and Compston (13) is closely similar to that of Swank. Alexander's scale (2), although presumably a more accurate measure of disease, is difficult to adapt to other scales, which refer chiefly to motor activities.

TABLE 1

*Approximate Equivalents Used for Comparing the Status of Patients as Reported by Different Authors**

McAlpine and Compston	Alexander	Kolb	Muller	Swank (Grade No.)
1		Working		1
			Not disabled	1.5
2	100			2
			Slightly disabled	2.5
3 and 4	200	Ambulant	Moderately disabled	3
5 and 6	260	Chair or bed	Severely disabled	4
		Dead		5 (Dead)

* Equivalent neurological grade numbers used by different investigators and their relationship to grade numbers used by Swank.

PATIENTS NOT ADHERING TO THE DIET

In analyzing the effects of a diet it is important to include only patients who adhered to the diet closely and constantly. On the evidence obtained by frequent reports from the patients, and discussions with them, it was concluded that of the 147 patients remaining, 29 should be rejected from the analysis for dietary reasons. In seven cases the reports of dieting were judged not reliable. Three cases failed to keep the diet from the start. Nineteen cases (14 per cent) of the remaining 137 were rejected because they discontinued the diet for long periods, or lost contact with us after a fair trial of dieting. Among these 19, it is important to distinguish those who abandoned the diet after some deterioration, and possibly because of it. Since the patients who do poorly on any treatment are those most likely to abandon it, their omission from analysis might introduce an appreciable bias favoring the treatment. Of these 19 cases six abandoned the diet while doing well. Hence, although their omission may cause a bias against the diet, it is unlikely to favor it. Six other patients were severely disabled from the start, and found the diet too difficult to follow. In one case we are unable to determine when the diet was abandoned. In the remaining six cases the failure to follow diet occurred after some deterioration.

All of the last 13 cases were in the "late phase" of the disease when they started the diet, and continued deterioration was not surprising. The omission of the last six cases

TABLE 2

*Details of Cases Starting Diet in 1948–1951**

No.	Years M.S. before diet	Start of diet	Age at start of diet	Relapses: Years before diet			Relapses: Years on diet										Grade Nos.						C	Activity	
				3	2	1	1	2	3	4	5	6	7	8	9	10	At start	1 year	June 1956	June 1957	June 1958	June 1959		At start	In 1959
17	0.08	12/49	26			2	0	0	0	0	0	0	0	0	0	0	2	1	1	0.5	0.5	0	+	F	F
2	0.08	6/51	21				1	0	1	0	0	0	0	0			2	2	2	2	2	2	NC	Amb.	F
10	0.08	10/50	33				0	0	0	0	0	0	0	0	0		1	1	1	1	1	1	+	F	F
43	0.08	9/50	46				0	0	1	0	1	1	0	0	0		2	1	1	1	1	1	+	Amb.	F
45	0.17	12/51	25				1	1	0	0	0	1	0	0			2	0.5	1	1	1	1	+	Amb.	F
82	0.17	4/51	21				2	0	1	0	1	0	1	0			3	3	3	3	3	3	NC	Amb.	Amb.
35	0.25	7/51	20				0	0	X	0	1	0	0	0			4	1	1	1	0.5	1	+	Bed	F
51	0.25	12/49	33			2	0	1	0	0	0	0	0	0	0	0	1	1	1	0.5	0.5	1	+	F	F
19	0.33	9/51	28				1	1	0	0	0	0	0	0			2	1	2	2	2	2	+	F	F
8	0.42	12/49	18			3	1	1	0	1	0	0	X	0	0	0	4	2	1	1	1	1	+	Ch.	F
91	0.50	8/49	36			2	X	0	0	0	0	0	0	0	0	0	3	3	3	3	3	3	+	Amb.	Amb.
4	0.60	1/51	32				0	0	0	0	0	0	0	0			2	1	1	1	1	1	+	F	F
5	0.60	9/49	27			2	0	0	0	0	0	0	0	0	0	0	1	0	1	0	0.5	0	+	F	F
113	0.92	12/50	33				0	0**	0	0	0	0	0	0	0		2	3	4	4	4	4	−	P	Ch.
18	1	10/50	23				1	0	1	0	1	0	0*	0	0		1	2	2	2	2	2	−	F	P
64	1.17	9/49	30		1	2	0	1*	0	0*	0	0*	0	0	0*	0	2	2	4	4	4	4	−	F	Bed
86	1.42	6/50	37		1	2																			
83	1.50	7/51	26				1*	0	0	0	0	0	0	0			2	3	4	4	4	4	−	Amb.	Ch.
34	1.50	11/50	39				0	0	0	0	0	0	0	0	0		1	1	1	1	1	1	+	F	F
28	1.60	10/51	26				0	0	0	X	X	0	0	X			2	1	0.5	0.5	0.5	1	+	F	F
102	1.92	12/48	41		3	2																			
56	2	7/50	29		1	1	1	0	0	1	0	0	0	0	0		2	1	0	0	0	0	+	F	F
93	2.50	12/49	41	0	0	3	X	1	0	1	0	0	0	1	0	0	4	3	3	2	2	2	+	Ch.	F
27	2.50	9/49	37	1	0	1																			
50	2.67	12/50	25				0	0	1	0	0	0	0	0	0		1	1	1	0.5	0.5	0.5	+	F	F
47	2.83	1/51	44				0	0	0	0	0	0	0	0			1	1	1	1	1	1	+	F	F
42	3	9/51	27				1	0	0	1	0	1	0	0			1	0	0	0	0	0	+	F	F
24	3	7/50	25	1	0	3	1	X	X	X	X	0	X	0			2	2	2	1.5	1	1	+	F	F
72	3	1/49	29	2	1	0	1	0	0	0	0	0	0	0	0	0	2	2	2	2	2	2	+	Amb.	Amb.
73	3	8/49	27	1	2	1	X	0	0	0	0	0	0	0	0	0	3	3	3	3	3	3	+	Amb.	Amb.
79	3	5/50	51	1	1	1	X	0	1	0	0	0	0	0	X		3	3	2	2	2	3	NC	Amb.	Amb.
84	3	9/49	39	1	1	1	0	0	0	0	1	0	0	0	0	0	2	1	1	1	2	2	+	F	F
23	3.25	1/52	25	1	1	1																			
145	3.60	1/49	30	2	1	1																			

145	3.60	1/49	30	2	1	1																				
52	4	5/51	25				1	0	0	0	0	0	0	1			2	1	1	1	1	1	+	Amb.	F
1	4.08	4/49	26	0	2	2	1	0	0	0	0	1	0	0	0	0	2	1	1	1	1	1	+	F	F
61	4.08	7/50	31	1	0	1	1	0	0	0	0	1	0	0	0		2	0.5	1	1	1	1	+	Amb.	F
38	4	5/49	32	0	1	2	0	1	0	0	0	0	0	0	0	0	1	1	1	1	1	1	+	F	F
46	4	1/51	24				0	0	0	0	0	0	0	0			2	1	1	1	1	1	+	F	F
135	4	9/51	44				0	0*	0	0	0	0	0	0			2	2	2	2	2	2	−	P	Amb.
44	4.92	4/50	46	1	1	1	0	0	0	0	0	0	0	0	0		2	2	1	1	1	1	+	P	F
121	4.92	5/50	34	1	0	2	0	0	0	0	0	0	0	0	0		4	4	4	4	4	4	−	Ch.	Ch.
32	5	10/51	22				0	1	1	1	0	0	0	0			2	0	1	1	1	2	−	F	F
54	5	12/48	37	1	0	1	X	0	X	0	0	0					2	1					+	P	
25	6	3/51	41				0	1	0	1	0	0	0	0			2	2	2	2	2	2	NC	F	F
40	6	4/50	40	1	0	1	0	0	0	0	0	0	0	0	0		1	1	1	1	1	1	+	F	F
66	6	5/50	23	0	0	2	1	1	0	0	0	0	1*	0	0		2	2	3	4	4	4	−	Amb.	Ch.
123	6	5/50	28	1	0	1	0	0	1	0	0	0*	0	0*	0		3	3	4	4	4	4	−	Amb.	Bed
128	6	12/51	29				0	0	0	0	0	0	0	0*			3	3	3	3	3	4	−	Amb.	Ch.
137	6	3/51	38				0*	0	0	0	0	0	0	0			3	3	4	4	4	4	−	Amb.	Ch.
143	6	5/51	30				0	0	0	0	0	0	0	0			3	3	3	3	3	3	+	Amb.	Amb.
125	6	12/48	34	0	2	2	1	0	0	0	0	0	0	0	0	0	4	4	3	4	4	4	−	Ch.	Ch.
92	7	5/51	26				0	0	1	0	0	0	0	0			2	2	2	1.5	1.5	2	+	F	F
155	7	7/51	38				0	0	1	0	0	1	0**	0			2	2	2	2	3	3	−	F	Amb.
85	8	4/50	22	2	2	3	0	0	X	0	0	1	1	0	0		1	1	1	1	0	0	+	P	F
104	8	1/49	42	3	3	1	1	1	1	0	0	0	0*	0	0	0	3	3	4	4	4	4	−	Amb.	Ch.
150	8	4/49	39	0	1	0	0	0	0	0	0	0	0	0	0	0	3	3	3	3	3	3	NC	Amb.	Amb.
75	9	3/50	31	1	1	0	0	0	1	0	0	0*	0	0	0*		2	2	2	2	2	3	−	F	Amb.
95	9	3/50	36	1	1	1	0	0	1	0	1	0	0				2	2	2				+	F	F
127	9	1/50	38	2	2	2	0	1	1	0	1*	0	0	0	0*		2	2	3	4	4	4	−	Amb.	Bed
129	9	7/51	45				0	0	0	0	0	0	0	0			3	3	3	2.5	2	3	−	Amb.	Amb.
68	12	8/49	48	0	0	1	0	X	0	0	X	0	0	0**	0	0	2	2	3	3	3	3	−	F	Amb.
154	12	4/51	45				1*	1*	1**	0	0	0	0	0			2	3	4	4	4	4	−	F	Bed
131	13	4/51	35				1	0	0	0	0	0*	0	0*			3	3	3	3	3	4	−	P	Ch.
76	14	11/49	33	0	0	0																			
69	14	12/48	43	1	1	1	X	XX	0	0	0	0	0	0	0	1	2	2	1	1	1	2	−	F	F
133	14	3/50	49	0	0	1	0	0	0	0	0*	0	0	0	0*		2	2	3	3	3	4	−	P	Ch.
116	14	4/49	39	1	0	1																			
112	16	1/51	32				1	0*	0	0	0	0	0	0			4	4	4	4	4	4	−	Ch.	Bed
98	17	7/49	44	0	0	0																			
105	17	4/51	48				0	0	0	0	0	0	0	0			2	3	3	3	3	3	−	Amb.	Amb.
106	19	1/49	39	0	0	0	0	1*	0	0	0	0	0	0	0	0*	4	4	4	4	4	5	−	Ch.	Dead
144	20	8/49	38	0	0	0																			
107	23	2/51	54				0	0	0	0	0	0	0	0			2	2	2	2	1	1	+	Amb.	F

TABLE 2—(*Continued*)

No.	Years M.S. before diet	Start of diet	Age at start of diet	Relapses: Years before diet 3	2	1	Relapses: Years on diet 1	2	3	4	5	6	7	8	9	10	Grade Nos.: At start	1 year	June 1956	June 1957	June 1958	June 1959	C	Activity: At start	in 1959
139	28	1/49	49	0	1	0																			
114	30	7/51	41				1	0*	0	0	0	0*	0	0			3	3	3	4	4	4	–	Amb.	Bed
Sum of relapses				27	31	56	22	14	15	6	7	7	3	2	0	1									
Number of patients				34	38	43	66	66	66	66	66	66	65	63	35	17									
Mean relapse rate for each year				0.795		1.30	0.33		0.23		0.106		0.046		0										
				0.817				0.21		0.091		0.106		0.032		0.059									

* X signifies an "off-diet" relapse.

The grade numbers prior to 1956 have been omitted except for the pre-diet number and the first year on diet. The number of years omitted can be found for any patient by subtracting the date of starting diet from June 1956, and deducting one from the number thus obtained. After the first year on diet, changes in grade were very slow, and when the 1956 value does differ from that given for the first year of diet, the changes in the intervening years can best be recorded as occurring by a uniform fraction in each year concerned.

The case numbers in column 1 are significant in showing an assessment of each patient's condition made soon after their first attendance. Numbers 1–60 were in an early phase of the disease, numbers 61–96 were difficult to assess but possibly advanced, and numbers 97–155 were already in a late phase of the disease when first seen.

Working activity: This is divided into 6 grades. Full-time work (F), part-time work (P), ambulant (Amb.), confined to wheelchair (Ch.), bedridden (Bed), and dead. To facilitate comparison of this series with others, the years in which downgrading in this scale of activity occurred are marked with an asterisk in the columns for relapses, each asterisk indicating a fall of one grade. These asterisks do not necessarily coincide with relapses, and in some cases (as number 106) denote progressive deterioration without noticeable "episodes."

The column headed "C" shows a plus sign for patients judged to have improved while on diet (up to 1959), "NC" for cases showing no noticeable change, and a minus sign for those showing deterioration.

may bias the results to a small extent for the 58 "late phase" cases, and it is possible that the omission of the six severely disabled cases, and of the one uncertain one listed above, may also bias the results in the same direction. Since all thirteen had considerable disability with an average duration of multiple sclerosis exceeding 10 years before starting the diet, this bias can have no effect on the results for cases treated in the early phase of the disease, and practically none on results for those treated within eight years of the onset of disease.

An appreciable bias against the treatment could be introduced by patients who were unwilling to confess to departures from the diet. This may have occurred in more cases than the seven mentioned above as unreliable. We believe, however, that gross prolonged infractions were detected.

Minor departures from the diet undoubtedly occurred, and relapses occurring at times when the patients had reported their consumption of saturated fat as double the allowance or more, for a period of two or more weeks, were recorded as "off diet" relapses. There were 28 of these in 896 patient-years, giving an "off-diet" relapse rate of 0.031. In Tables 2 and 3 they have been indicated by the letter X. As experience in dealing with the patients and their diets increased, the number of these relapses per patient-year fell from 0.04 for cases starting diet in 1948–1951, to 0.016 for those starting in 1952–1954.

INITIAL CONDITION OF CASES

For the 118 cases who adhered to the diet, the average grade number at the start was 2.16. They included 10 cases (8.5 per cent) in grade 4. Sixty-two cases (52.5 per cent) were initially working and walking, suggesting that in this series the disease was slightly more advanced than in that of MacLean and Berkson (14), where 70 per cent of the patients were initially working and walking.

USE OF "CONTROL" CASES

The most effective control in a study of treatment of multiple sclerosis would involve giving alternant cases no treatment or the best alternative treatment. This procedure is only ethical in a prolonged study of multiple sclerosis if the physician considers both treatments of equal value. It is only practicable if the patients think the same; otherwise the patients will be quick to note differences and they will promptly try to adopt the more favored treatment.

An unreliable method of assessing treatment of a remitting disease is the comparison of identical patients before and after only one or two years of treatment. Patients with multiple sclerosis tend to come to a clinic soon after a relapse, when remissions are to be expected even if no treatment is given. Hence comparisons made over brief periods introduce a strong bias into a study, and their use has led many authors to advocate specific treatments which have later been abandoned as useless. This type of error can be greatly reduced by certain precautions which are discussed later in this paper.

Results may be assessed with fair reliability either by comparing a series of treated cases with untreated patients in the same series, using these precautions, or by comparing series published by different authors with full regard to variables such as prior duration of disease and the initial severity of cases.

In making comparisons with cases of other authors we have found the chief obstacle to be the general use of summarized tables which give inadequately detailed data about individual patients. Outstanding exceptions are the informative tables given by Compston (8) and Allison (4). In Tables 2 and 3 we have therefore given the chief data needed for comparing the present series with others. From these details many useful analyses can be made.

TABLE 3

*Details of Cases Starting Diet in 1952–1954**

No.	Years M.S. before diet	Start of diet	Age at start of diet	Relapses: Years on diet 1	2	3	4	5	6	7	Grade Nos.: At start	1 year	June 1956	June 1957	June 1958	June 1959	C	Activity: At start	In 1959
30	0.08	3/53	43	1	0	0	0	0	0		3	1	1	1	1	1	+	Amb.	F
36	0.08	5/53	24	0	0	0	0	0	0		2	0	0	0	0	0	+	Amb.	F
7	0.08	6/53	50	0	0	0	0	0	0		4	2	1	1	1	1	+	Bed	F
16	0.08	3/54	26	1	0	0	0	0			1	1	1	0.5	0	0	+	F	F
37	0.08	3/53	42	0	0	0	0	0	0		2	1	1	1	1	1	+	Amb.	F
148	0.17	11/53	32	0	0	0	0	0	0		3	3	3	2	2	2	+	Amb.	P
12	0.25	10/53	30	0	0	0	0	0	0		1	1	1	0.5	0.5	1	+	F	F
49	0.25	2/52	25	1	0	0	0	0	0	0	2	1	1	0.5	0.5	0.5	+	Amb.	F
97	0.33	3/53	33	0	0	0	1***				3	3	3	5				Amb.	Dead
14	0.33	11/53	24	0	0	1	0	0	0		2	1	1	0	0	0	+	F	F
39	0.67	5/53	24	0	0	0	0	0	0		3	2	3	3	3	3	−	Amb.	Amb.
152	0.67	9/53	20	1	1*	0	0	0	X*		4	4	4	4	4	5	−	Ch.	Dead
33	0.83	1/53	28	0	0	0	0	0	0		1	1	1	0.5	0.5	0.5	+	F	F
94	0.92	1/52	28	0	1	0	1	0	0	0	2	2	2	2	2	2	−	F	F
21	1	7/53	23	0	0	0	1	0	0		1	1	0	1	1	1	+	F	F
146	1	7/53	43	1	0	1	0	0	0		3	3	3	3	3	3	−	Amb.	Amb.
59	1.17	4/52	26	0	1	0	0	0	0	0	1	1	0	0	0	0	+	F	F
6	1.17	1/53	20	1	0	0	0	0	0		0	0	0	0.5	0.5	0	+	F	F
3	1.42	11/53	23	0	0	0	0	0	0		2	1	0	0	0	0	+	Amb.	F
74	1.60	9/52	36	2	0	1	0	0	0	0	2	2	2	2	2	2	+	Amb.	P
55	2	12/52	25	1	0	0	X	0*	1*	0	1	1	2	2	3	3	−	F	Amb.
88	2.17	10/52	34	0	0	0	1*	0	1	0	2	2	2	2	2	2	−	F	P
10A	3	5/53	26	0	0	0	0	0	0		1	0	0	0	0	0	+	F	F
23	3.25	1/52	25	0	1	0	0	0	0	0	1	0	0	0	0	0	+	F	F
26	3.33	5/53	23	0	0	0	0	0	0		2	0	1	0	0	0	+	F	F
134	3.42	1/53	25	0	0	0	0	0	0		3	2	2	2	2	2	+	Amb.	P
40A	3.50	6/53	30	X	0	0	0	0	0		1	1	1	1	1	0	+	F	F
58	3.50	6/52	28	0	1	0	0	0	0	0	1	0	0	0	0	0	+	F	F

41	4	3/52	45	0	0	0	0	0	0	0	1	1	1	0.5	0.5	0.5	+	F	F
65	4	9/53	24	1	1	0	0	0	0		2	3	3	3	3	3	−	Amb.	Amb.
120	4	7/53	43	0	1	0**	0	0	0		3	3	4	4	4	4	−	P	Ch.
132	4	10/53	28	0	0	0	0	0	0***		2	2	3	3	3	5	−	Amb.	Dead
20	4.17	3/53	25	0	0	0	0	0	0		1	0	0	0	0	0	+	F	F
70	4.25	4/52	30	1	0	0	0	0	0	1	2	2	2	2	2	2	−	Amb.	P
9	4.50	12/53	28	1	0	0	X	0	0		1	1	1	1	1	2	−	F	F
130	4.67	3/52	28	0	0	0	0	0	0	0	4	4	4	4	4	4	−	Ch.	Ch.
90	5	5/52	28	0	0	0	0	0	0	0	2	2	2	1	1	2	+	Amb.	F
13	6	11/53	32	0	0	0	X	0			0	0	0	0	0	0	+	F	F
57	6	4/54	30	0	0	0	0	0			3	2	2	2	2	2	+	Amb.	F
60	6	5/53	49	0	0	0	0	0	0		1	1	1	0.5	0	0	+	F	F
103	8	11/53	39	0	0	0	0*	0	0		2	2	2	2	3	3	−	P	Amb.
48	10	12/53	51	0	0	1	0	0	0		2	1	1	1	1	1	NC	F	F
119	10	7/53	30	0	0	0	0	0	0		3	3	3	3	3	3	NC	Amb.	Amb.
126	10	5/52	35	1	0	0	0	0	0	0	3	3	3	3	3	3	−	Amb.	Amb.
11	12	10/53	33	0	0	0	0	0	0		2	1	1	1	1	1	+	F	F
71	12	7/53	46	0	0	0	0	0	0		2	2	1	1	1	2	NC	P	F
101	12	5/53	40	0	0	0	0	0	0		3	3	3	3	3	3	NC	P	P
78	14	12/52	35	0	1	0	1	0	0	0	3	2	2	2	2	2	+	Amb.	P
151	14	3/54	42	0	1	0	0	0			3	3	3	3	2	1	+	Amb.	F
136	19	10/53	45	0	0	0	0	0	0		2	2	2	2	2	2	NC	P	P
153	20	2/52	47	0	1*	0	1*	0	0	0	3	3	4	4	4	4	−	P	Ch.
111	22	6/53	54	0	0	0	0	0	0***		3	3	3	3	3	5	−	Amb.	Dead
Sum of relapses				13	10	4	6	0	2	1									
Number of patients				52	52	52	52	51	46	15									
Mean relapse rate for each year of dieting				0.25	0.193	0.077	0.115	0	0.0435	0.067									

* See Table 2 for description.

RESULTS

The comparison of "control" cases with cases treated by Swank's diet will be discussed under three main headings: frequency of relapses, changes in neurological grades, and changes in working ability.

FREQUENCY OF RELAPSES IN UNTREATED CASES

When comparing relapse rates, four variables need special attention. There is, first, the tendency of patients to forget minor relapses; second, the tendency of patients to seek treatment first soon after a relapse; third, the variable borderline between "relapses" and "fluctuations" as defined by different observers; and fourth, the time interval between any relapse and the onset of disease.

1. TENDENCY OF PATIENTS TO FORGET MINOR RELAPSES

Many patients tend to forget minor health incidents a few months after they occur, and become unable to distinguish minor relapses from fluctuations. The observer must keep in mind that the mildest attacks will be the most easily forgotten.

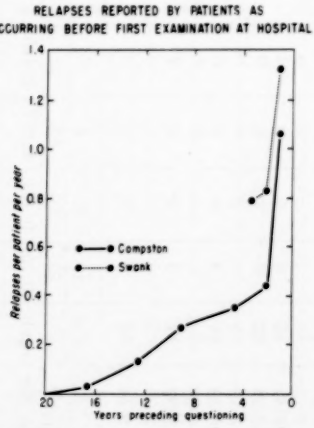

FIG. 1. Relapse rates reported by patients when questioned as to their past history. The dashed line shows the rates for 43 of Swank's patients as reported for the three years prior to questioning. The continuous line shows the rate for Compston's patients as reported for periods up to 20 years before questioning.

This tendency is illustrated in Figure 1, which shows the relapse rates reported by Compston's patients (8) for the years prior to their examination. From the second to the 20th year before questioning, the rate falls fairly steadily by an amount averaging 0.025 relapses for each year. On the assumption that the relapse rate does not alter with duration of the disease, this decrease suggests a measure of the patient's forgetfulness.

When estimating relapse rates at different periods of the disease by taking histories, this forgetfulness can cause two types of error. First, the incomplete recall of earlier events by longstanding cases reduces the rates found for their earlier years, and consequently lowers the average relapse rate for the entire period. Second, patients giving a long history will limit the number of minor relapses reported for any one period, and will recall chiefly the severe relapses. On the other hand, histories covering periods of only two to three years will tend to include minor attacks almost as completely as severe ones. Imperfect memory will also influence the extent to which a group of episodes occurring in fairly quick succession is reckoned as a single relapse, or as several. If recorded as the events occur, or soon after, such a series of episodes may well appear as multiple attacks, whereas five years later they will be recalled as a single event.

2. TENDENCY OF PATIENTS TO FIRST SEEK TREATMENT SOON AFTER A RELAPSE

This feature was emphasized by Compston (8) and is also illustrated in Figure 1. In both curves the relapse rate is much higher for the year immediately before admission to the clinic than for the preceding years. The apparent increase in relapse rate from the second to the first year before admission is approximately 0.5 for Swank's series, and 0.6 for Compston's. This sharp increase in the apparent relapse rate the year before treatment causes serious error if the relapse

rate for only a few pre-treatment years is compared with the rate while on treatment. This error can be avoided by Compston's practice of omitting the year immediately before treatment when computing relapse rates before treatment, although this procedure probably lowers the apparent rate below the true average, and adds a small error due to an extra year of forgetfulness.

If the natural distribution of relapses tends to be periodic rather than random, large errors may occur when comparing rates before and after treatment, unless a sufficient number of years are included in both groups to cover more than one complete relapse cycle. Picture a regular cycle of one relapse in every third year, giving the following sequence:

Z T

1,0,0,1,0,0,1,0,0,1,0,0,1,0,0,1,0,0,1,0,0.

If an ineffective treatment is begun in the year marked "T," it is evident that a correct comparison of pre-treatment and post-treatment years will result if the year marked "Z" is omitted and three, six or nine years on each side of it are used for comparison. The treatment will then appear to be without effect. If, again omitting year "Z," we average relapses for three years before "T" and only two years after beginning treatment, the treatment will appear to have stopped relapses entirely. If all four years before treatment are compared with the first four years of treatment the rates will appear to be 0.5 before treatment and 0.25 during it. While relapses are not as regularly periodic as this, the schema does illustrate the danger of relying upon short study periods.

In some patients relapses do tend to come in groups rather than at regular or wholly random intervals. It is evident that if these patients start treatment preferentially during the first half of a group of relapses, they will show higher relapse rates during treatment than before it, suggesting that the treatment is harmful. The converse holds if they tend to start treatment towards the end of a group of relapses.

When comparing relapse rates before and during treatment, it seems best to use as many years of treatment as possible, and certainly not less than three. Of the pre-treatment years, that immediately before treatment may well be omitted, and only the previous two, or possibly three, years be included. The onset or first attack should then, we think, be counted as a relapse if coming within the period concerned, and a correction should be made for the *rate of forgetting* if that can be estimated. Even with these precautions, the danger of errors from grouped relapses must dictate caution unless the treatment has continued for at least six or seven years.

The optimum duration of the pre-treatment period used for comparison may also be influenced by the relapse rate for the geographic area under consideration. If the normal rate is high the optimum length of the observation period will be relatively short. If the normal rate is low, the observation period should be longer in order to include adequate numbers of relapses and to reduce error if there are periodic variations in rate.

3. VARYING DEFINITION OF "RELAPSE" AND "FLUCTUATION"

It is possible that different authors have used significantly different criteria for recognition of relapses. Thus McAlpine and Compston (13) in an especially intensive study of 189 patients, recorded 38 out of 309 "episodes" as lasting less than one day. Relapses as brief as this are most unlikely to be recorded in hospital notes referring to periods of time some years before the questioning, as used in Muller's series. Further, some investigators would probably designate a day-long episode as a fluctuation rather than an exacerbation, especially if it coincided with some minor infection or severe psychological source of disturbance.

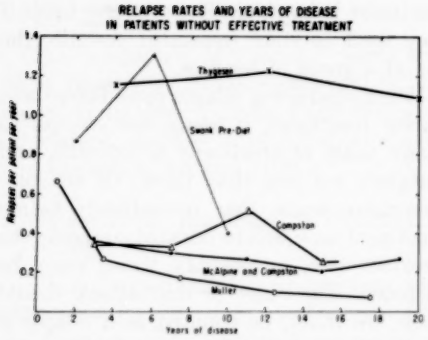

FIG. 2. Relapse rates plotted against duration of disease, as reported by different authors for patients without effective treatment. Except in Muller's series, the rates found by any one observer decrease only slightly between the second and eighth year of disease. To smooth the curves, the data have been averaged over periods of disease of eight years for Thygesen's figures and of four years for those of Swank, of Compston, and of McAlpine and Compston.

4. RELAPSE RATES AND DURATION OF DISEASE

Figure 2 shows relapse or "attack" rates as recorded by various authors, plotted against duration of multiple sclerosis. The onset is excluded except in Swank's cases. The data for Thygesen are taken from Figure 2 of his monograph (24). They were obtained by questioning 58 patients at intervals which averaged three weeks during the 18 month period of observation. Of the 105 attacks which Thygesen recorded only six were the direct cause of inclusion of a case within the observed series. No correction has been made for these six attacks. Thygesen excluded monosymptomatic cases and those totally incapable of walking. The dashed line shows the attacks occurring in the second and third years prior to first attendance for 37 patients studied carefully by Swank (20). The small number concerned makes the curve irregular, but it suggests that no great fall in relapse rate occurs before the eighth year of disease, although after that a rapid decrease occurs in this series. Compston's data (8) refer to 85 patients attending a London hospital for the first time between September, 1947 and 1949. They include only those relapses occurring from the second to the sixth year before attendance.

The curve for McAlpine and Compston (13) is derived from 393 patients seen in London, twice yearly from April, 1948 to September, 1950, (with an average duration of disease of about seven years before 1948) and from case notes covering earlier periods. Muller's data are derived from his monograph (16), using his Table 22, which shows calculated probabilities derived from data grouped to avoid bias in the longstanding cases. They are based on a single interview with patients or their relatives at a time when the average duration of disease was about 14 years, and upon hospital notes extending back for many years. These notes were first recorded when the average duration of disease was 6.1 years (the median was just over three years). Under these conditions it seems probable, as Muller suggests, that more "bouts" occurred than were recorded.

The average values for each of these five curves show considerable inverse correlation between their relapse rates and the length of time between the occurrence of the relapses and their recording. This may be due chiefly to the factor of forgetfulness discussed above, plus the tendency of routine hospital notes to mention only the major relapses. It may be of importance that both Compston's cases and those of McAlpine and Compston were studied after about eight years of severe restriction of the British diet caused by war-time rationing. This factor, which is discussed later, may account for their low relapse rates.

PRACTICAL CONCLUSIONS

With the exception of Muller's data which are in this case suspect, the curves in Figure 2 show no major fall in relapse rate between the first and eighth year of disease. The mean relapse rate for the first eight years or more seems unlikely to be less than 0.4 per patient year in Western Europe and North America, and in countries with a high stand-

ard of nutrition is probably near 0.75, the value found by Alexander (3) for 554 of his cases. It is possible, as found by Thygesen (24), that an even higher rate prevails where greater care is used in the taking of patients' histories.

RELAPSE RATES IN PATIENTS ON DIET

Figure 3 shows the relapse rates during successive years on the diet for Swank's 118 patients. The upper line marked with crosses includes all patients who, in general, continued to practice the diet. The lower continuous line shows the rather smaller rates found when those relapses were excluded which had occurred at times when patients were "off diet" for two weeks or more. The dashed line shows an exponential curve plotted to the equation:

$$\text{relapse rate} = 0.4083(0.7273)^t$$

where "t" is the number of years on diet as plotted. The exponential form is of theoretical interest as being consistent with any gradual reparative process when the rate of repair at any time is proportional to the amount of damage then present. The damaged tissues could be abnormal lipids in myelin as discussed by Sinclair (17), or abnormal blood vessels as suggested by changes in the blood-brain barrier shown by Broman (6). The rise in the tenth year appears appreciable on the graph, but is due to only one mild relapse in one of the 17 patients who had completed 10 years on the diet.

Figure 3 shows that even in the first year on diet, the relapse rates are much lower than they were before dieting (Figure 2), and that in subsequent years the rate continued to fall, nearly halving its value every two years. From the second year onward these rates fall much below any of those in Figure 2, except Muller's. Owing to the steep fall of Muller's curve, a proper comparison with Swank's data can only be made for corresponding years of disease. A simple way of doing this is to consider only those

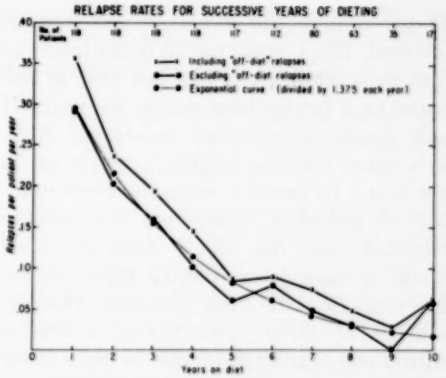

FIG. 3. Relapse rates in successive years for patients on low-fat diet, averaged for patients in all stages of the disease. The upper curve includes relapses occurring in patients when "off diet." The dotted line is an exponential curve approximately fitting the lower continuous line which shows relapse rates in patients on diet. The mean relapse rates for 43 of these patients during the three years preceding dieting was 0.90, or for the second and third year before diet 0.80. The numbers just below the upper horizontal boundary of the graph indicate the number of patients available for each point on the graph.

of Swank's cases for which dieting began within two years of the onset of disease (averaging approximately one year). Then successive years of diet such as 1, 2, 3, correspond to the years of disease, 2, 3, 4, and so on. These cases from Swank's series (39 in number), show the following relapse rates for successive years of disease when fully on diet, starting with the second disease year: 0.41, 0.21, 0.18, 0.10, 0.11, 0.054, 0.044, 0, 0, 0. These figures are all much below Muller's rates for the corresponding years of multiple sclerosis (second to 11th year). If "off diet" relapses are included, the figures are still lower than Muller's, especially after the first three years on diet.

Swank's results can also be compared with Muller's Table 23, which shows the frequency of occurrence of a new bout within one year of a preceding one. In Muller's series this frequency falls slowly with successive bouts, so that while 48 per cent of the first bouts (onset of the disease) were followed by a further bout within one year,

43 per cent of the seventh bouts were so followed. His average for all bouts from the first to seventh gives 46.1 per cent as followed by a further bout within one year. (If only remittent bouts are considered, Muller's figure remains roughly constant at 54 per cent.) In Swank's series the exact number of preceding bouts was not usually recorded, but can rarely have exceeded seven in cases where dieting began within five years from the onset of disease. The percentage of these cases having a relapse within one year while on diet is much lower than Muller's series. It averages 16.2 per cent for all the relapses, and only 9.2 per cent after the first to second year on diet. These figures include "off-diet" relapses. They are considerably lower if these are omitted, or if only cases starting diet in 1952–1954 are included.

If the rapid fall in relapse rates found for patients on diet is in fact due to the diet, it is of great importance. It therefore seems desirable to examine other factors which may possibly have influenced the results.

1. EFFECT OF DURATION OF DISEASE

The evidence given above, that relapse rates in untreated cases do not fall rapidly between the second and eighth year of the disease, is strengthened by the data shown in Figure 4. This illustrates the relation between relapse rates and duration of disease for patients after fixed periods on diet (omitting "off-diet" relapses). In the first year of diet the relapse rate does fall as the duration of disease rises from two to about eight years. But for the later years of diet no definite trend is visible, at least from the fourth to the 16th year of multiple sclerosis.[4]

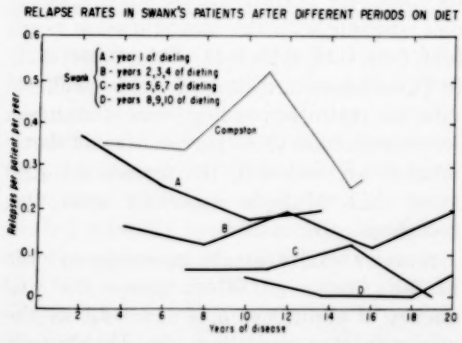

FIG. 4. Relapse rates plotted against years of disease for groups of patients after different periods on diet. For comparison the figures for Compston's series from Figure 2 are shown. In each line the data have been averaged over periods of four years of disease.

In contrast to this small influence of the duration of disease on the relapse rate, the change at any given year of multiple sclerosis with successive years on diet is obvious, in spite of the irregularities caused by small numbers. *It seems safe to conclude that the fall in relapse rates shown in Figure 3 is not due to the increasing years of multiple sclerosis which accompany the increasing years on diet.*

2. PSYCHOLOGICAL EFFECTS

An important factor in experimental therapy is the interest of the patient, and especially his desire to please the investigator by showing a favorable response to treatment. Patients with multiple sclerosis are especially sensitive to encouragement and prone to subjective improvement. Usually they have received little attention from their physician, their condition being regarded as both uninteresting and hopeless. The misleading effect of encouragement, however, is of short duration. In earlier studies on vasodilating drugs, Swank[5] found that most patients reacted favorably for one or two months, but that after three to four months almost all patients developed a neutral attitude toward the drugs. We therefore have

[4] Since no patient began diet before the onset of multiple sclerosis, there are no data for the earliest years of multiple sclerosis in the later years of diet. Hence in averaging three years of diet together, the first points for the second, fifth, and eighth years of diet had to be omitted to avoid bias. If the figures for individual years are studied, they suggest a slight excess of relapses in the first four or five years of multiple sclerosis, but this may only be due to random variation in small numbers. After the 14th year of multiple sclerosis the numbers are too small to have significance.

[5] Unpublished observations.

little fear of errors from this cause after the first year or two on the diet.

Another possible source of error lies in the decreased frequency of contact with the patients which occurred in the course of the test. Prior to January, 1954, the patients were seen at monthly intervals during their first year on low-fat diet and thereafter at intervals of six weeks to three months. After this date they were seen once a year in June, except in 1954, when they were examined in April. Since the observer's move to Portland in April, 1954, the diet has been checked by mail four times a year in most cases, and by interview once a year. Telephone contact became negligible. Since a high standard, involving the reporting of minor relapses was achieved in the earlier years, it is possible that the number of relapses reported may have fallen later, either from forgetfulness due to decreased contact, or from an increased tendency of the patients to disregard small changes in their condition. The forgetting rate of 0.025 per year shown for Compston's cases in Figure 1 would have a negligible effect on results reported at 12 week intervals, but the most trivial relapses which certain observers would term fluctuations, might be forgotten.

It is also possible that as patients become accustomed to the disease, their tendency to report minor fluctuations as definite relapses, decreases. A check on both these sources of error was made by an analysis of the changes in relapse frequencies with successive calendar years. This showed that for patients in any given year of diet, there was little decrease in the relapse rate for those starting diet in 1948–1951. For those starting diet in 1952–1954, however, there was a definite decrease in the rate after 1954. The greatest percentage decline occurred after 1956. However, data for the 1952–1954 cases are influenced by the change to a more stringent diet, and by varying percentages of cases in the late phase of the disease. It appears that the above sources of error may have contributed to a decrease in the reporting of the smallest relapses, but cannot possibly account for the paucity of major relapses. It seems unlikely that a serious error was thus introduced.

3. Relation between frequency of relapses and deterioration of patients' condition

Since steadily progressive cases are generally believed to fare worse than those which relapse and remit, it is of interest to check if frequent relapses did indicate a poor prognosis. Swank's results favor this view, since 26 patients deteriorating by one or more grades while on diet had 39 relapses (1.5 per case), while 44 patients who improved by similar amounts while on diet had only 31 relapses (0.706 per case). Thus deteriorating patients averaged more than twice as many relapses as those improving. This tendency was shown in all phases of the disease.

Changes in neurological status

Figure 5 shows grade numbers on Swank's scale as defined above, plotted against years of disease. The dashed line shows Swank's cases before diet. The continuous lines show the same series of patients after starting the diet at various intervals from the onset of

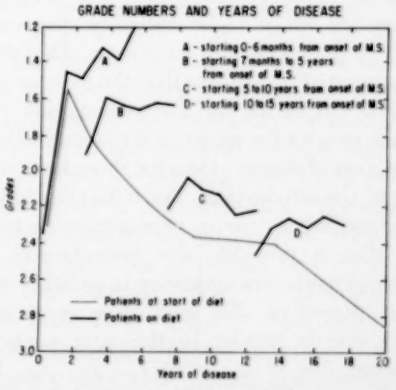

FIG. 5. Average numbers plotted against years of disease. The dashed line shows patients before dieting, and the continuous lines show cases which started diet at various intervals after the onset of disease. The number of patients in the four groups were: A = 21; B = 52; C = 24; and D = 13.

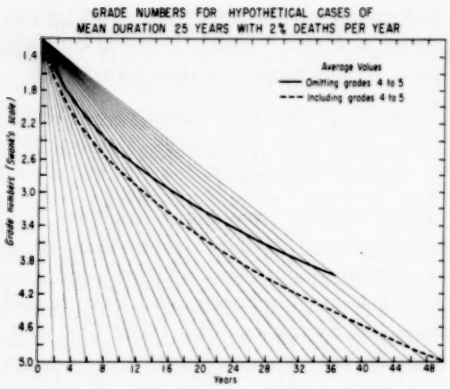

FIG. 6. Chart showing a simple method of estimating the average grade numbers to be expected in a representative sample of patients with multiple sclerosis. In this example the mean duration to death is assumed to be 25 years after first recognition of the disease at grade 1.2. The straight lines indicate a uniform deterioration to death at grade 5 at rates such that two per cent of cases die in each year after onset. The lower curve shows the average grade numbers to be expected under these conditions, and the upper curve the average expected in a clinic which excludes wheelchair and bedridden cases.

multiple sclerosis. The following conclusions seem justified.

The initial rise in the dashed curve is probably due to a tendency for cases with a severe onset to visit a clinic within the first year of disease, while those with a milder onset are likely to be correctly diagnosed only in their second or later years. This view is supported by the fact that the average improvement during the first year on diet was much greater for patients first seen in their first year of disease, than for those first coming in the second year. The initial rise in the four continuous curves representing patients on diet is probably due very largely to natural remissions occurring in patients who were placed on diet soon after an attack. This rise is smaller in the later years of disease.

Curve A shows a definite improvement in neurological status continuing for five years after starting the diet. Curve B rises initially and then remains roughly level. Both separate further from the pre-diet curve as the treatment continues. The continuous curves are plotted only for the first five years of dieting because in the later years differences in the numbers of subgroups concerned make this method of averaging unreliable. Since the persistence of apparent benefit to patients after the first year of diet is of great interest, a more accurate study is illustrated in Figures 6, 7, and 8.

Figure 6 shows a simple graphical construction useful for finding the average grade numbers to be expected under any specified condition. Here neurological grade numbers are plotted against years of disease. The straight lines show the course of hypothetical cases with an average duration to death of 25 years, and a uniform death rate of 2 per cent for each year of disease. These values are derived from various sources, including McLean and Berkson's death rate of 20 per cent in the first 10 years (14), Kurland and Westlund's median life expectance of 22.6 years (12), and an extrapolation of the death rate from Muller's Tables 36 and XV which points to 50 per cent of deaths at 28 years from onset. The initial value of 1.2 is chosen as fitting Swank's pre-diet cases after remission from the first attack. The assumption of a uniform time interval for each unit of grading is based on a rough estimate of the average clinical course, and agrees with Allison's finding (4) that in fatal cases about half the total period of disease is spent in a state of "severe incapacity." In Figure 6 the lower curve (dashed line) is produced by averaging the grade numbers thus delineated for any given year of disease. The upper curve is similar except for the elimination of all cases in grades 4 and 5. The difference between these curves is small for the first 16 years or so, showing that the omission of bedridden or wheelchair cases from any series has less effect than might be expected. It is also of interest that at 25 years, the highest grade shown, that for a patient having uniform descent to death in 50 years, is as low as 3.1, while the average for all cases is still 3.85.

GRADE NUMBERS OF CONTROL CASES

Figure 7 shows similar curves presenting the results of various authors, which have been translated to Swank's scale as shown in Table 1. It includes for comparison the uppermost straight line and the upper curve from Figure 6. Allison's 40 cases (4) fall roughly in the center of this group, but are omitted to avoid overcrowding. The line for Alexander's 554 cases is estimated from the trend of the points shown in Figure 3 of his paper (3), since the equation there given denotes an undue prolongation of the later phases of the disease. Muller's data are calculated from Tables 39 and 44 of his monograph. The values for Kolb are estimated from measurements of Figure 5 in his paper (11).

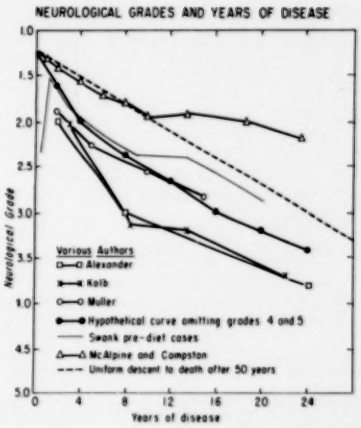

FIG. 7. Average neurological grades plotted against years of disease, as found by different investigators, but converted to a common scale. For comparison the uppermost straight line and the upper curve from Figure 6 are included.

All the curves based on clinical studies resemble the hypothetical curve in showing a reduced slope after the first few years. Their average slope for the years shown (excluding the later years of McAlpine and Compston), ranges from 0.088 to 0.071 grades per year, a closer agreement than would be expected from the rough approximations involved in comparing the scales of different authors. The average at 0.075 is close to the slope of 0.076 shown by the hypothetical straight line for death at 50 years. The curve for McAlpine and Compston, however, becomes abnormally flat after 10 years, and if projected, would reach grade 4 only at 94 years and grade 5 at 135 years. This flattening is mainly due to the omission of the 30 severe and 78 fatal cases mentioned by the authors. The high level and gentle slope of the earlier part of their curve fits well with their low relapse rates and may possibly be related to the 10 years of rationing experienced by these British patients. This rationing caused a considerable reduction in intake of fat and sugar during and after the 1939–1945 war (9, 10).

It seems safe to conclude that Swank's pre-diet cases resemble those of other authors sufficiently to use as "controls," although the small numbers of patients plotted for the last two points of this curve (11 and 10 patients, respectively) render these two points unreliable.

Table 4 gives data facilitating the plotting of "control" curves, including the hypothetical curves, Swank's pre-diet curve and the average of the five observers' curves in Figure 7, but omitting the data of McAlpine and Compston after the first eight years of the disease. The observers' curve is best suited for comparison with cases on first arrival at a clinic, while for series which include deaths and bedridden patients, the last line in the table is preferable.

In Figure 8 the uncertainties of averaging which restricted the data used for Figure 5 are avoided by plotting only three points which could be compared for all of Swank's patients, namely the grades observed before dieting, after the first year, and after the last year observed. These data include every case from Tables 2 and 3. The continuous lines summarize *changes* in grades for all Swank's cases while on diet, and distinguish groups with different pre-diet intervals. All three groups show improvement (reduction in grade number) during the first year on diet. The earliest group, A, shows continuing improvement during subsequent years,

TABLE 4

*Grade Numbers During Course of Multiple Sclerosis in "Control" Cases**

	Years from Onset of Disease								
	0	2	4	8	12	16	20	24	50
Average of studies by Alexander, Kolb, Muller, and Swank and the first 8 years of McAlpine & Compston		1.76	2.04	2.51	2.85	3.04	3.31		
Swank (pre-diet)		1.65	1.95	2.32	2.39	2.57	2.86	(3.15)	
Hypothetical average omitting grades 4 and 5	1.20	1.65	1.96	2.37	2.67	3.00	3.21	3.40	
Hypothetical including all cases	1.20	1.78	2.09	2.57	2.95	3.26	3.54	3.79	5.00

* Grade numbers during course of multiple sclerosis in "control" cases.

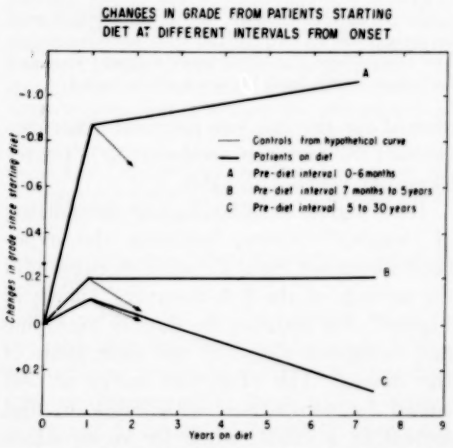

FIG. 8. Average *changes* in neurological grades for all patients while on diet, divided into three groups starting diet at different intervals from onset of disease. The continuous lines show only the changes during the first year on diet, and those found at the last year observed for each patient (usually 1959), after from 5 to 10½ years of dieting. The dotted lines show the rate of fall of the hypothetical curve (shown in Figure 6) at times from onset of disease equal to those of Swank's cases after one year on the diet.

while group B shows little further change and group C shows a decided fall. For comparison the dashed lines show the rate of fall of a control curve from points calculated to have the same prior duration of disease as Swank's cases had after their first year on diet. Since the latter include several deaths and a number of patients in grade 4, the control curve chosen is the lower of the two in Figure 6 (the last line in Table 4). In each of the three groups it is evident that the control series depreciates faster than the dieted series, although in group C the difference is much too small to be significant.

We think that the improvement due to a natural remission occurring shortly before a patient's first visit to a clinic will have little effect on the clinical course of disease in the second and later years of dieting. Certainly natural remissions will not account for the widening gap between control curves and those for patients on diet as the period on diet lengthens (as seen in curves A and B in Figures 5 and 8). These results therefore fit well with the reduction in relapse rates already described, and strengthen the probability that the dieting cases have received some real benefit, at least for those who started dieting within five years of the onset of disease. The small or uncertain benefit in group C (Figure 8) is to be expected, since patients with many preexisting lesions would seem likely to continue to deteriorate even if given a treatment so successful that it prevented formation of new lesions.

This analysis in terms of the interval between onset of disease and the beginning of dieting supplements a previous study (22) based on the severity of disease at the start of dieting. That study, which is further developed in a monograph (18), showed much

better results in cases starting diet in the "early" or slightly disabled phase than in those starting diet when the disease was already advanced.

DEATHS

Among these 118 cases five deaths occurred before the last examination in June 1959, at four, six, nine, 28 and 29 years after the onset of disease. From the 1959 examination until April 1960, one further death occurred in case number 154, after 21 years of disease. All six deaths occurred in patients who had been assessed as in the "late" or advanced phase of the disease when first placed on the diet.

The overall death rate of 6/118 (5.1 per cent) in eight years compares favorably with Millar's figures (15) from a follow-up study of cases attending a hospital in North Ireland. He found death rates for a period of five to nine years of 24 per cent in polysymptomatic cases and 13 per cent in monosymptomatic ones. Practically all of Swank's cases were polysymptomatic. Hence, Millar's cases had a death rate about four times greater than Swank's cases. Abb and Schaltenbrand (1), in a five-year follow-up study of 958 cases attending the Wurzburg clinic since 1935, found that 240 had died (25.1 per cent). For those who had attended the clinic during the first five years of disease, he found the still higher death rate of 36.5 per cent (65 out of 179 patients). For the corresponding 73 patients in Swank's series the death rate during eight years on diet was 4.12 per cent, or about one-eighth the rate found by Abb and Schaltenbrand in the shorter period of five years.

These figures emphasize the wide variations in death rates for patients attending different clinics. While social and economic factors may account for many of the differences, especially in cases in the later stages of the disease, the difference in mortality rates for patients attending the clinic for the first time during the first five years of the disease suggests some additional factor. It seems possible that diet, either before or after the onset of disease, was partly responsible for this difference.

COMPARISON OF TWO DIETS

As already mentioned, patients starting the diet in 1952–1954 were allowed less saturated fat, and prescribed more unsaturated fat than those who had started in 1948–1951. The results for the 1952–1954 group were consistently better, showing lower relapse rates and greater neurological improvement while on diet.

The difference in relapse rates is considerable; a chi-square test for the proportions relapsing between the second and the fifth years on diet giving $.02 < p < .05$. Similarly, the proportion of relapses followed by a further relapse within one year is much reduced in the 1952–1954 group, as mentioned above.

It seems quite possible that these differences are due to the use of a more stringent diet in 1952–1954, but other possible causes, such as random variations or changing conditions in Montreal, cannot be excluded with certainty.

CHANGES IN WORKING ABILITY

While changes in working ability are only a crude measure of disease, since they are influenced by social and economic conditions, they have, in Swank's series, proved closely correlated with changes in neurological status. It therefore seems legitimate to use them for comparison with the results of other workers. MacLean and Berkson (14) report the survival rate of ability to work and to walk of 278 cases at Rochester who were able to work and walk at their first visit to the clinic. They suggest that this group furnishes "a minimal base line that a proposed therapeutic regimen must improve on before evidence for effectiveness can be accepted." An approximate comparison of their series with Swank's is shown in Figure 9. It appears that the percentage of cases deteriorating in Swank's series was less than

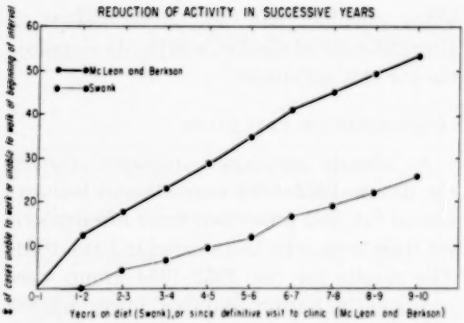

FIG. 9. The upper curve shows the percentage of patients reported by MacLean and Berkson as becoming unable to work or unable to walk in successive years from first attendance at the Rochester clinic. The lower curve shows the corresponding figures for Swank's patients while on diet, calculated by the actuarial method used by MacLean and Berkson. At year 0 all patients in both series could walk and work.

half that in the Rochester series. While the two series are roughly comparable, a strict comparison is not possible because we do not know the mean interval between onset of disease and arrival at the clinic for the 278 cases included in MacLean and Berkson's series. For the entire group of 418 patients from which these 278 were selected, this interval averaged 7.0 years, while for Swank's entire series the interval averaged 6.5 years. The selection of only those cases who could walk and work on first attendance must have reduced this interval in the Rochester series, and in Swank's lowered it to 5.07 years for the 62 cases, all of the remitting type, which are plotted in Figure 9.

An increase in prior duration of disease of 0.5 years for Swank's cases would have a somewhat similar effect to moving Swank's curve in Figure 9 half a year to the left. This would only slightly reduce the difference between the two curves.

It seems probable that the difference between Swank's series and the Rochester one is due to benefit from the diet, although differing degrees of social and economic stimulation may have influenced the results.

DISCUSSION

When judged by three criteria the modified low-fat diet appears to have benefitted patients with multiple sclerosis. First, its use was followed by a rapid reduction in relapse rates to very low values after about four years on the diet. Second, there followed an improvement in the average neurological status which persisted for the duration of the test in patients who began the diet within five years from the onset of the disease. Patients who began the diet after disease had been present longer than five years did not experience an average improvement in their neurological status, but possibly deteriorated more slowly than controls. Third, when compared with the series reported by MacLean and Berkson, a much higher percentage of patients on low-fat diet were able to continue working and walking for periods up to 10 years from onset.

In addition to the results analyzed statistically in this paper, it is to be noted that relapses of multiple sclerosis became progressively less severe and prolonged in patients on diet (20–22). It can also be added that relapses rarely attacked previously non-involved parts of the nervous system in patients on diet longer than one year. These last two changes in the course of the disease were probably important.

The effects just discussed seemed to have a cumulative influence on the course of the disease. Although definite advantages appear to have been derived by the patients in the first year, progressive lessening of relapse rates, and improvement in the performance of many of the patients through the entire test period were observed.

A further change noticed in many patients was a cumulative decrease in fatigue, evident after the first two to three years on the diet. They became more energetic, active, and productive. This improvement was most evident in patients who had had the disease for less than five years when placed on diet.

While these results accord well with observations correlating the geographic distribution of multiple sclerosis with the quantity of fat in local diets (19), there is ample evidence to show that an excess of dietary fat in adult life is not the sole factor causing multiple sclerosis. Not merely do many people eat large quantities of fat without incurring this disease, but even on the low-fat diet five of the 48 patients who started it within three years of onset of disease deteriorated by two or more grades. It is impossible to state whether this deterioration was due to changes in pre-existing lesions, or failure of the change in diet to stop the formation of fresh lesions. A further discussion of the factors involved is given in a recent monograph by Swank (18).

Further evidence of the need for postulating one or more additional factors in the initiation of the disease is suggested by the observation of McAlpine and Compston (13), in a careful study of the diet of 50 cases of multiple sclerosis. They found "no specific deficiency" and no increase in fat intake during the period preceding onset of multiple sclerosis, and "the diet not differing from that recommended for sedentary workers in general."

This observation is, however, of limited weight for a number of reasons. First, the cases were studied after nearly 10 years of rationing which involved considerable reductions in the consumption of fats and sugar for most people, and as Drummond (9) has also commented, led to a much greater equality of distribution of diet than had occurred in pre-war times. Second, if the onset of a disease is governed by two or more factors, its incidence among individuals in any particular population may be governed by the amount of any one of the factors present in those individuals, and need not be proportional to the quantities of each factor present. Third, Brickner and Brill (5) found the diet of many of their cases of multiple sclerosis deficient, especially in fat. Swank (18) noted that many patients with this disease seemed to eat less fat than normal and progressively less as the disease advanced. This may be a sign of defective lipid metabolism in these patients (whether conditioned by lack of physical exercise or other more fundamental factors), so would account for the observations of McAlpine and Compston. We conclude that, while adult diet is not the sole factor concerned in the disease, the results with Swank's series are sufficiently definite to justify treatment with a low-fat diet as used in this study. Since the low-fat diet is most effective early in the disease, treatment should be started in its earliest recognizable phase, and even in some cases while the diagnosis is still uncertain.

Since the selection of alternant patients within one series for use as controls is either not practical or to be avoided, there is a great need for effective comparison between the different series of cases. Such comparisons as we have shown in this paper were restricted to very few series because most authors have published only summarized tables which do not supply the data needed for effective comparisons. The necessary data include the date of onset of the disease; the grade numbers estimated on some simple scale of motor ability such as those of McAlpine and Compston, or of Swank; the dates at which grade numbers or changes in them were observed; and, in series where observation was adequate, the number of relapses in each year with estimates of their severity and duration. Given these data, together with normal information about the selection of cases, it would be possible to compare different series closely. Even small series of a dozen patients would be useful if the observations extended over, say, four years or more. There must be many thousands of case notes from which such data could be abstracted, with the resultant production of a reliable control series that would be invaluable for further studies of treatment, or for studies of geographic vari-

ations in the severity and course of the disease.

SUMMARY

One hundred and eighteen cases of multiple sclerosis have been studied for from five to ten and one-half years while on a diet low in total lipids with 50 per cent or more of these consisting of vegetable or fish oils. The results are compared with those published in other studies of this disease and appear to show that the diet is a definite improvement on conventional methods of treatment, particularly if started within five years of onset of disease.

Attention is called to the possibility of obtaining improved control series for investigations of this disease if, instead of the customary summarized tables, certain relevant details are made available.

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THE RELATION OF TIME OF LIFE TO BEHAVIORAL CONSEQUENCE IN BRAIN DAMAGE: II. THE ORGANIZATION OF TACTUAL FORM EXPERIENCE IN BRAIN-INJURED ADULTS[1]

HERBERT G. BIRCH, M.D., PH.D.[2] AND LILLIAN BELMONT, PH.D.[1]

INTRODUCTION

The present report is the second of a series concerned with contrasting the consequences of brain damage sustained in childhood and in later maturity. Underlying these investigations is the hypothesis that, together with locus of injury, extent of neurologic damage and type of lesion, the time in the life history of the individual at which damage is sustained is a significant variable in the determination of functional consequence. Available information on this question is still both sparse and indirect (4), despite the fact that attention was directed to the significance of the time of life at which injury was sustained almost twenty years ago (3).

In part, our lack of information with respect to the differential consequences induced by the timing of brain lesions in the life history is a function of the difficulty inherent in defining populations of individuals at different ages with lesions of the nervous system which are comparable in all respects other than time of occurrence. Although studies may be conducted in a controlled way at the animal level, clearly such a line of inquiry is morally enjoined at the level of human investigation. An alternative procedure is possible, however, if one proceeds from the premise that brain injury with respect to locus, extent, type, etc. tends to be unsystematically distributed at any given age level. Once this assumption is made, it becomes possible, at least in a preliminary way, to examine the effects on behavioral functions of brain injury sustained at one time of life with brain injury sustained at a later stage of development by comparing the differences between each group of brain-injured persons and its own normal age-mate controls. When this is done, the differences between the normal and brain-injured subjects at each age level may be compared as induced differences. Such a comparison, then, is not a direct comparison between groups injured at different life stages but is one step removed. It consists of the consideration of any discrepancy between the differences exhibited by each of the brain-damaged age groups when compared with its respective control. This method we have called the method of *pseudo-comparison* (1).

In our earlier report (1) we found differences in performance on a series of visual reconstructive tasks by brain-injured children and brain-injured geriatric patients. Whereas brain-injured children were markedly inferior in their performance to controls, far less stereotypy and less definitive difference appeared when the brain-injured adults were contrasted with neurologically intact older people.

Since the earlier study involved a largely visual and intellective task, a fuller understanding of the problem can be achieved only if tasks involving other sensory and perceptual systems are explored. The present report, therefore, derives from our analysis of changes in tactual recognition and discrimination by brain-injured adults and children.

METHOD

The subjects directly examined in this study were 40 adult patients on the re-

[1] This study was conducted at the Bird S. Coler Hospital in the Department of Physical Medicine and Rehabilitation, New York Medical College, New York, N. Y.

[2] Present address: Department of Pediatrics, Albert Einstein College of Medicine, New York, N. Y.

habilitation wards of a large municipal hospital for chronic diseases. Twenty of the patients were hemiplegics, brain-damaged as a result of cerebral vascular accidents (CVA) and 20 were non-brain-damaged, but orthopedically handicapped. The orthopedic control group contained seven leg amputees, five arthritics, four fractures of the lower extremity, and four with miscellaneous minor orthopedic complaints. The subjects were selected on a random basis from a master file containing the records of all patients currently receiving treatment on the rehabilitation service. The brain-damaged patients used in the study were between 50 and 70 years of age at the time of damage. In all cases at least six months had elapsed since the original injury, and no severe aphasia, no hemianopsia, and no uncorrected visual or hearing defects were present. The non-brain-damaged patients were matched to the brain-injured group for age and social background. The one discrepancy between the two groups was in the sex distribution. There were eight men and 12 women in the brain-damaged group as compared with 14 men and six women in the control group.[3]

The tactual discrimination task (Werner's tactual-motor test, 5) is designed to elicit disturbance in tactual figure-background discrimination by means of introducing background interference. The task consists of two sets of form boards. Set I consists of three thumbtack-covered boards with raised semi-spherical tacks outlining the forms (square, oval, triangle) to be perceived tactually. Set II consists of three boards which contain the identical forms reproduced as raised, wooden solids against a flat, smooth wooden background. Figure 1 is a schematic drawing of the forms used in Set I and Set II.

The materials were presented in a cloth-covered box so that the subject could feel

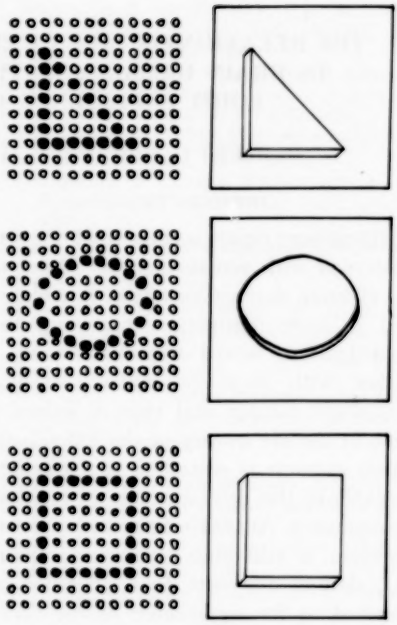

FIG. 1. Tactual stimuli.[4]

but could not see the boards. The handedness of the patient was determined before the task was presented. Hemiplegic patients discriminated with the intact hand, whether or not it was habitually preferred in the premorbid state. Control subjects used the preferred hand in their tactual discrimination. No time limit was used.

The following instructions were given to the subject:

"Under this cloth is a board with a lot of things arranged in a design. I am going to put your hand on it and I want you to feel it and then draw the design. Be sure to remember the design because I shall ask you to draw it."

Set I was presented, followed by a rest period of five minutes. Set II was then presented with the following instructions:

"Now here is another board. I'd like you

[3] Analysis of performances in relation to sex of subject revealed that sex difference did not significantly affect group performance.

[4] These stimuli are exact reproductions of those used by Werner and Strauss in their studies of figure-ground disturbance in childhood brain-damage.

to feel it and then draw a picture of the design."

After each board was presented, the subject was asked to draw a picture of what he had felt. Multiple-choice items were constructed for use with two patients who had great difficulty in drawing. The use of multiple-choice items in these two was an assurance that motor inability *per se* was not confused with perceptual dysfunction in the event of failure in accurate reproduction (2).

In order to evaluate the subjects' behavior on the tactual form boards, it was necessary to codify the findings. Categories for analysis were established to facilitate comparison with the analysis of behavior alteration observed by Werner and Strauss in their studies of children with early brain damage (5). Certain additional categories emerged from the adult data and were also used in analysis.

The Set I responses of the adult subjects were classified into the following categories:

A. Background Dominance
   1. *Background response only:* placement of circles and dots (to represent background tacks) without any form drawn.
   2. *Background response—incorrect form:* placement of circles and dots (background influence) with an incorrect form drawn.
   3. *Background response—correct form:* placement of circles and dots (background influence) with the correct form drawn.

B. Figure Dominance
   1. *Inaccurate form response:* no response to the background stimuli (tacks) but inaccurate form perception, with no relation to the objective structure of the presented stimulus.
   2. *Close form response:* no response to the background and a line drawing in which the overall aspect corresponds to a feature of the presented stimulus (*e.g.* drawing a circle instead of an oval).
   3. *Accurate form response:* recognition of and/or correct reproduction of the geometric form presented.

On Set II, the adult subjects' responses were scored "right" or "wrong." A form was considered incorrect under two conditions: 1) if the subject had already demonstrated the ability to make accurate reproductions and then drew an incorrect form on Set II; or 2) if the wrong multiple choice item was chosen.

For the purpose of a comparison with the data on children, the accuracy measure of the subject was ignored since Werner and Strauss (5) classified their subjects according to: 1) responses to background, 2) responses to the background and the form, or 3) responses to the form on Set I. It was reported by them that none of their children, brain-damaged or control, had difficulty with the forms on Set II (forms presented against a flat, smooth background in which circumstance background influence is at a minimal level of effectiveness).

RESULTS

The data presented in Tables 1 and 2 make it clear that significant differences exist between the adult brain-damaged group and the adult control group with respect to (1) the accuracy of form response

TABLE 1

Difference in Accuracy in Tactual Discrimination for Adult Brain-Damaged and Control Groups

Category	Brain-Damaged (N = 20)		Control (N = 20)		t	p*
	Mean	S.D.	Mean	S.D.		
Number of correct designs per subject on Set I	0.5	1.0	1.45	1.3	2.63	<0.01
Number of correct designs per subject on Set II	2.35	0.7	2.9	0.3	3.06	<0.005

* One-tailed test.

TABLE 2

The Effect of Background on Tactual Perception in Adult Brain-Damaged and Control Groups

Group	Percentages of Responses with Background Interference*		t	p†
	Mean	S.D.		
Brain-Damaged (N = 19)	79	37.2	2.23	<0.03
Control (N = 20)	50	44.0		

* Responses showing background interference were determined by comparing responses on Set I (with background) with the corresponding responses on Set II (no background). Responses to Set I which were poorer than the corresponding response to Set II were counted as showing background interference. One subject in the brain-damaged group was omitted because no interpretable drawings were produced on either Set I or Set II.

† One-tailed test.

and (2) the degree of disruption induced by background interference. In all comparisons it is found that the adult control group performed more adequately. The data indicate that the brain-injured patients were significantly less accurate in their reproduction of tactually experienced designs whether or not background interference had been operative.

The brain-injured patients made more pure background responses than did their controls. The number of subjects in the brain-injured group who showed pure background responses was greater than the number found in the control group and approaches statistical significance ($p = .10$ by chi-square comparison). When the number of designs with background contamination was related to the number of designs in which pure figure responses were made, once again it was found that the brain-injured adults were subject to greater background influence than were the control subjects.

However, the effect of background interference is best analyzed by a comparative consideration of Set I and Set II responses. Therefore, background interference is determined in Table 2 by a comparison of responses on Set I, which involve a markedly interfering thumbtack background matrix, with the responses to Set II, where the smooth wooden surface provides minimal interference with contour and so may be considered as baseline or neutral background context.

In Table 2 the degree of background effect manifested in the performances of the brain-damaged and control groups is compared. It may be seen that background interference is significantly greater in the brain-injured patients.

Thus far, the analysis has concerned itself entirely with the differences in the capacity to reproduce or to visually recognize tactually perceived materials on the part of our brain-injured adults and their orthopedically handicapped but neurologically intact controls. The brain-injured group was found: (1) to be perceptually inaccurate and to make significantly more errors in reproducing the geometric forms than did the controls, and (2) to be more influenced than the controls by characteristics of tactual background.

In order to explore the relation of the performance of our patients to those of children, data were obtained from the report of Werner and Strauss (5) on children's responses to the tactual discrimination tasks. Upon inspection of Table 3 it appears that the differences in performance between the brain-injured children and their controls are more pronounced than the differences which were obtained by comparing brain-damaged older adults with their neurologically intact controls. Whereas brain-injured adults exhibited some increased degree of background interference, in children with brain damage background interference occurred ten times more frequently than in children without such injury. It appears that differences in the behavior of the respective control groups is the source for the marked difference in the

degree to which brain damage results in deviant visual reproduction of tactual perceptions in children as contrasted with older adults. Both brain-damaged groups showed more deficit than did the respective controls, but, the effect of brain injury in childhood apparently resulted in more deviant performance than did adult injury.

DISCUSSION

The most striking difference found in the comparison of our data with those of Werner appears to lie in the relatively higher level of background interference induced by early childhood brain damage. This difference, however, appears to be a function of differences in the performance of the control groups rather than a true difference between the two brain-injured samples. Older adults with no known injury tended to exhibit a much more marked susceptibility to tactual background influences than did the control group of children.

Several possibilities may be advanced to account for this difference. One hypothesis is that, developmentally, tactual perception in the normal adult differs from that of the normal child by attempting to be more inclusive and to incorporate both figure and ground in the reproduction of a tactual experience. This possibility is given little support in our data, since frequency of background incorporation in the controls is not significantly elevated. Further, an independent supplementary test of a small series of six younger adults (age range: 30 to 40) failed to reveal any instance of background influence.

A second possibility derives from a consideration of the clinical characteristics of the adult and child brain-injured populations. The adult brain-injured individuals were all hemiplegics whereas the brain-injured children showed no gross motor handicap. It is possible that more focalized damage exists in our adult experimental group and more diffuse distribution of injury in the children.

TABLE 3

The Effect of Brain Injury on Background Influence in the Tactual Perception of Brain-Injured Children and Brain-Injured Adults

Type of Response	Percentage of Responses					
	Children*			Adults		
	Brain-Damaged (N = 25)	Control (N = 25)	Difference between B-D and Control	Brain-Damaged (N = 20)	Control (N = 20)	Difference between B-D and Control
Background	53	4	+49	58	40	+18
Background and Form	21	3	+18	10	7	+3
Form Only	26	93	−67	32	53	−21

* The data for the children were obtained from the published findings of Werner and Strauss (5).

Since none of the adult control subjects had a motor disability which interfered with accurate drawing, the simplest explanation for differences in our control population as with Werner's control children, musculo-skeletal disorder interfering with design drawing, must be rejected. However, if one considers the possibility of diffuse neurologic impairment with aging, it is possible that background influence occurs in our control population as a function of diffusely distributed senile brain change despite the absence of localizing neurologic signs.

SUMMARY AND CONCLUSIONS

The behavior of a group of 20 hemiplegic patients and a group of 20 non-brain-damaged patients with musculo-skeletal disorders were compared on a tactual reproduction task. It was found that the adult brain-damaged group showed increased deficit in tactual reproduction marked by inaccuracy and increased background interference.

The responses of the adults were then compared qualitatively with the performances on the same task by a group of brain-damaged children and a control group of their peers as reported by Werner and

Strauss. It was noted that the brain-damaged children showed greater comparative deviance from controls in tactual reproduction than did the adult brain-injured group. These results are discussed in relation to the problems of aging and of locus of brain-injury.

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PSYCHOANALYSIS AND SCIENTIFIC METHOD

LAWRENCE S. KUBIE, M.D.[1]

INTRODUCTION

Psychoanalysis remains a center of various recurring controversies. Of these, its place among the sciences is perhaps the most crucial. If we are to assay this objectively, we must recognize not only its present limitations but also its potentials for growth and for future research. Automatically this focuses our attention on psychoanalysis as a process of exploration and not on the superstructure of psychoanalytic theories or the various forms through which these have evolved or their deviants. Most emphatically, psychoanalysis will not be evaluated here as a therapeutic instrument. There are compelling reasons why attempts which have been made to compare the therapeutic efficacy of any or all of the psychotherapies have been premature and culpably naive. The technical problems which must be solved before such studies can be meaningful have been discussed repeatedly in papers to which the interested reader will be referred.

These studies underscore the fact that ever since 1930 (when the Berlin Institute published its honest but overconfident report on the apparent therapeutic fruits of the first ten years of its existence), psychoanalysts have devoted much thought to the problems involved in the evaluation of therapy. In spite of these efforts, for the reasons explained in these publications, none of these evaluative studies has yielded significantly informative data. It is not surprising therefore to find that those who fail to appreciate the complex difficulties of such studies and the even greater difficulty of comparative evaluations of different psychotherapies blunder even more seriously in their efforts. Indeed, most studies of this problem bear the imprints both of naivete and of bias.

Although unavoidable, for reasons to be summarized below, it is regrettable that psychoanalysis had to begin as a therapeutic technique. Its development would have been sounder had it been possible for it to start as an instrument and object of basic research, unbiased by therapeutic urgencies. This is not the first time in medical history that new agents and new procedures have been applied prematurely to therapeutic purposes, and then in some measure have been misused even by their discoverers and proponents. Nor is it the first time that this has given rise to misinterpretations and misunderstandings. This has been true of many drugs, of many biological agents such as sera and antibiotics, and of many physical agents such as heat, cold, electrical stimulation, the x-ray and other forms of radiant energy. Such errors are always corrected gradually.

It is misleading to assume, as some do, that mercenary reasons account for the early use of psychoanalysis for therapeutic purposes. The deeper reason for this was the simple fact that at the outset only those who were in dire need and for whom existing therapies had brought no relief would subject themselves to this new, untried and arduous regimen. But for the existence of this vast pool of uncured and largely untreated neuroses, there could have been no psychoanalytic Odyssey. Moreover, these urgent appeals for help still tend to draw any humane investigator away from his researches into therapeutic involvement, unless the institution for which he works not only forbids him to assume therapeutic responsibilities, but also provides a pool of therapists to whom he can refer those whose needs become apparent to him in the course of his studies. Yet there are no full-time

[1] The Sheppard and Enoch Pratt Hospital, Towson 4, Maryland.

research institutes which have so far-sighted and humane a program as this.

As a science, psychoanalysis has paid a price for the fact that its origin and development was made possible only by its immediate commitment to the service of human needs. The price has been high; but it will not be too high in the long view of history, if we now set out on the process of correcting the consequent errors. Just because it was inevitable that anlysis was tossed into the therapeutic arena prematurely does not justify a continued tolerance of this today, when men and women are eager to undergo analysis for training purposes, as part of research projects, and out of sincere philosophical and intellectual curiosity. Indeed this fact points to a healthy change in the temper of our culture: a lessened defensiveness, a willingness to accept the complexity and the motley color of human personality, and a greater humility about the role of conscious processes in personality. This change is itself a result of the cultural impact of psychoanalysis, and has occurred in spite of its many scientific deficiencies.

A transparent error in logic resulted from the fact that human beings used to subject themselves to analysis only when driven to it by the failure of other forms of therapy, and because they had heard that somehow in the course of analysis a certain undetermined number seemed to reach a resolution of some of their difficulties. Hope and their own needs led them quite naturally to assume that in those cases in which therapeutic results had attended or followed analysis, the analytic experience had played a major role in these improvements. This of course is *post hoc* reasoning. In many instances it may have been true: but it remains unproven. For us as scientists such a sequence of events provides only a challenging working hypothesis, the testing of which is urgently necessary in spite of the difficulties with which such testing is beset. Yet one may wonder whether the earlier analysts would have had the courage to push ahead on their ever-lengthening and more arduous analytic voyages without some measure of innocent and optimistic lapses in logic. For this, too, we should in retrospect have tolerance and sympathy, as we now begin to apply more exacting criteria. In short, as we look back historically and with the detachment which is supposed to be an attribute of the philosophic mind, the fact that analysis began as a therapeutic instrument should lead us to conclude that insofar as this made possible the birth of psychoanalysis it was in balance good, even if it clouded many scientific issues. Now, however, we have reached a point at which we must use analysis both as an object of basic research and also as one instrument among many for the study of the analytic process itself. In fact in many centers this is starting already, gropingly, fumblingly, but with dedicated purpose. I do not doubt that the outcome will be many important changes in technique and in the superstructure of concept, terminology and theory.

In his original article in the *Encyclopedia Britannica* (10) Freud predicted that in the retrospective view of history, the cultural significance of psychoanalysis would be measured in terms of its value as a method of psychological investigation and as a theory of human psychology, rather than as a technique of therapy. Moreover, he warned repeatedly against the biases, both subtle and gross, which are introduced into analytic theorizing by an over-emphasis on therapeutic needs. Yet conscientious exhortations and self-correction cannot alone eliminate all of the distortions which flow from the therapeutic applications of psychoanalysis. Instead, we must find out how to subject the analytic process itself to direct investigation *outside* of therapeutic settings. Such a change, however, creates new technical problems and new fallacies. Much has been written about the differences which the absence of a therapeutic need and motive will introduce: but there are as yet no objective studies of these hypothetical if

reasonable assumptions. To explore them will be one of the first necessary steps towards a systematic research into psychoanalysis.

This is not all that can be said about the problems of research into the psychotherapeutic and psychonoxious leverage of psychoanalysis; but perhaps it is enough to indicate both some of its complexities, and also why I choose *not* to become involved in them until we can first survey the position of psychoanalysis in science from a nontherapeutic point of view.[2]

Therefore I will now consider in sequence (a) some of the inherent limitations of psychoanalytic methods; (b) the essence of psychoanalysis as a process; (c) how this process fits into the framework of scientific methodology; and (d) finally what we might call the philosophy underlying the concept of psychodynamics in general.

[2] The many extrinsic variables which must be taken into account in any effort to compare the results of various organic and psychological methods of treating psychiatric conditions were discussed in several symposia. One of these was held in Boston in the late forties and was published in the International Journal of Psychoanalysis in 1948 (40). Another was published in the American Journal of Orthopsychiatry in 1948 (4) and in 1949 (5). A statistical study of variations in psychoanalytic practice was published in Psychiatry in 1950 (18). The Hixon Fund Lectures (20), delivered at the California Institute of Technology and published by the Stanford University Press in 1952, considered the special problems of validation and prediction and the peculiar difficulties of setting up control groups in the psychotherapies. An article entitled "Some unsolved problems of psychoanalytic psychotherapy" (27), appeared in the volume called *Progress in Psychotherapy*, edited by Frieda Fromm-Reichmann and published in 1956 by Grune and Stratton. This paper considered various other factors which make such comparative studies premature at this time, and outlined the specific conditions which must be met before such studies can have any value. A description of the structure of a multi-disciplinary institute for basic research in psychiatry (26) was published in the Bulletin of the Menninger Clinic in 1956. A paper on "The neurotic process as the focus of physiological and psychoanalytic research," delivered in London in September of 1958 and published in the Journal of Mental Science, London (31), brought together still more of these factors. Finally, the difficulty of attaining the quality of technical, clinical and personal maturity which such research necessitates has been explored in still other studies (21, 31, 32).

LIMITATIONS

This will not be an exhaustive list of limitations. Nor will it be organized systematically. It is instead a representative sample of some of the limits within which psychoanalysis has operated in the past, and beyond which it is now trying to reach.

(1). Among the scientific disciplines and methodologies, psychoanalysis has had to struggle with unique difficulties which are consequences of the fact that it has been dependent largely upon auditory data, with only minor visual additions. Because of the dominant importance and speed of the spoken word as an instrument of communication, because of the slower pace of writing, and because of the limited range of variations and the consequent relative stereotype of gesture and expression as methods of communication, the ear has been the major source of psychoanalytic data. To an even greater extent than visual data, auditory data are vulnerable to distortion both in the moment of perception and in recall. As Abraham Kaplan pointed out, science has been forced to abandon the doctrine of immaculate perception (28).

Recent studies of subliminal and preconscious perceptions (most of which unfortunately have been restricted to tachistoscopic studies of visual input) have shown that the incessant bombardment of the individual by preconscious afferents plays a more important role than could have been appreciated by Freud. Indeed there is abundant evidence from daily experience that subliminal afferents from auditory, olfactory, tactile, kinaesthetic, proprioceptive and enteroceptive modalities play a major role in everything which goes on in the central nervous system (9, 15, 35). Yet even to this day their full implications for psychoanalysis have not been explored. Future research will have to include investigations of the processes of preconscious perception *via* every sen-

sory modality, and also of the processes of preconscious response. The purpose of this will be not only to free the psychoanalytic interview from its too exclusive dependence upon conscious auditory input, but also to include in its theoretical structure and in its exploratory and therapeutic techniques considerations of the role of preconscious (subliminal) input from all modalities, preconscious central processing, and preconscious responses on autonomic and somatomuscular levels.

Distortions of auditory data occur in a quite specific way during the very process of perception. As we listen to words, we alter what we hear. We do this "preconsciously," *i.e.* automatically and without being aware of it. In fact, this is essential both for successful cognitive communication and for empathic communication as well. We "correct" what we hear in our effort to understand what anyone says to us and how anyone feels in our presence. We do this as we listen to patients and again as we try to reconstruct from memory both what was said and how. To demonstrate this preconscious "correcting" of experience one need only record a sample of one's own free speech, and then listen to the recording while reading a transcript made by an expert secretary (32). One discovers that in transcribing from the machine, the conscientious secretary will have "corrected" what the tape said. Moreover, unless especially schooled not to do this, the better the secretary the greater will be the effort to have the tape "make sense," and the greater are the "corrective" distortions which will have been imposed upon the transcript of the recording. We can hardly escape concluding that until it became feasible to introduce techniques of precise recording and reproducing, the raw analytic data from analytic interviews rested on the insecure grounds of man's fallible memory of his distorted percepts. Nor was it possible to subject linguistic, para-linguistic or kinesic responses to formal or statistical analysis, until the techniques of recording and screening became generally available (32).

No one will deny that new distortions are produced by the introduction of mechanical contrivances into any highly-charged human relationship. There are comparable problems, however, in every science. The microscope and the stethoscope introduce distortions. Blood drawn from the vein is changed when it is transferred to a test tube for chemical analysis. If science advances in spite of these technical flaws it is by learning to recognize the changes that are introduced by the refined instruments of observation, of recording and of measurement, then by learning how to limit these changes and how to maintain their approximate constancy, how to allow for them, and finally how to correct for them. All of this must be done before it is safe to derive new theories from the data which new methods provide. Thus analysis is following a sound precedent as it prepares for the first time to make more precise records of the raw data of analytic communications.

Nor is it an accident that Freud's most basic research into dream psychology, which consisted primarily of his studies of his own dreams, were dependent not on anyone's spoken word, but on his own *written* records both of his own dreams and of his own free associations to them. Many years ago this fact alone should have alerted analysts to the importance of the permanent record for the advance of their own science.

(2). The fact that every moment of behavior is transitory forces us to depend upon memory, with its inevitable retrospective distortions. Moreoever, because that transient moment leaves its permanent trace on the observed and the observer, it can never be re-enacted unchanged. Therefore until there were ways to record and reproduce the material of psychiatric and psychoanalytic interviews, it was impossible for the observer to study them again and again, or to subject them to scrutiny by other observers.

(3). Another major limitation has been

the difficulty of setting up precise and adequate controls. This is a complex subject; but because it has been discussed in several of the publications to which I have already referred (27) the only point which it is necessary to make here is that the use of groups of supposedly similar ailments to balance against hypothetical normals is so fallacious as to make a mockery of the principle of control. Instead we are forced to use a more cumbersome and less precise method of control, in which each individual is studied in minute detail before any generalization is carried over from one to another. This method, however, introduces its own sources of error. Probably the answer will ultimately be found in developing methods of pooling the experience of many observers on many patients, each of which is recorded meticulously for parallel interpretation and statistical analysis. (A limited start has been made towards this by the Central Fact Gathering Committee of the American Psychoanalytic Association.)

The superficial study of large numbers has certain values; but it loses much of this value unless out of the larger group an unweighted and statistically adequate sample of representative individuals can then be subjected to microscopic scrutiny, by methods which are derived from formal analytic techniques. Again these steps introduce new errors; but they have at the same time their special values. Whether the values counterbalance the errors will require detailed testing.

(4). There are limitations in our techniques of *validation*. In the first place, although we speak of "an" interpretation, the determinants of human behavior are always multiple. As a result the word "interpretation" actually covers a group of interrelated interpretations. It has often been pointed out that it is not difficult to prove that certain interpretations of the meanings of behavior are possible (14, 44, 45). Nor is it difficult to take a step beyond that, and to prove that in many instances an interpretation is plausible or even probable. What is difficult is the critical final step, *i.e.*, to prove that in any specific instance any one interpretation is adequate and unique, and therefore constitutes the one necessary hypothesis. Psychoanalysis has rarely been able to present data which meets these ultimate criteria: yet this degree of evidence is of utmost importance for the validation of analytic procedure and techniques. Until we reach this point, any interpretation must be presented as being only one likely hypothesis among several—never as *the* interpretation.

Conclusive validation of a specific interpretation in a specific situation has been possible chiefly with the material from uninformed children, naive psychotic patients, and subjects under hyponosis (20). The literature is full of examples drawn from these three sources, where the patient, child, psychotic, primitive, or hypnotic subject sounds as though he had written the textbooks. These data carry the validity of precise experimentation (22, 31).

(5). In the past, validation has been approximated by applying several criteria: the emotional responses to an interpretation; the changes which occur in the patient's subsequent communications (*i.e.* changes both in form and content); concomitant changes in his stream of free associations; the sudden accessibility of hitherto inaccessible material (*i.e.* memories, feelings, conflicts, ancient fears and shames); still other changes which seem to follow upon the increased accessibility of such data. While these are by no means to be dismissed as meaningless, they are peculiarly liable to the *post hoc-propter hoc* fallacy. We must not forget the fact that in presenting an interpretation one frequently must talk perforce about some taboo topic. This in and of itself can free the patient to talk about this and/or related taboo topics, whether the specific interpretation was correct or incorrect. Therefore, a subsequent increase of "freedom" cannot be used as conclusive evidence for the validity of the specific interpretation.

As evidence, therefore, any changes in the material after an interpretation are circumstantial and inferential rather than conclusive. The only conclusive evidence of the validity of an interpretation would be if through it one achieved greater precision of predictions; and before this can be estimated, a vast amount of precise quantitative and qualitative work must be done to establish the degree of precision of predictions and the relative incidence of correct and incorrect predictions in settings which are freed from the pressing demands either of therapy or of teaching.

(6). A great deal has been claimed for the importance of *prediction* in the validation of interpretations. There is no doubt that in the daily practice of analysis, and particularly in the supervision of students, detailed and accurate predictions are occasionally made as to what is going to occur in the life of a patient—the kind of material that he is going to produce, the kind of dream which he is going to have, the kind of feelings that he is going to generate. Although sometimes such predictions are quite startlingly correct, no study has been made of the frequency with which such predictions are incorrect, nor of the ratio of the correct to incorrect predictions. Nor has anyone studied how frequently *no* predictions are made, which would be essential in a statistical survey. It is possible that the analyst becomes sufficiently confident to venture a prediction only under some special circumstances; and he may fail to venture a prediction out of a preconscious fear of being incorrect. As a result of a withholding of predictions, such predictions as are ventured clearly would not constitute a random or representative sample; and not to include the withholding would unfairly weight any sample of correct predictions in the favor of analysis.

Equally significant is the fact that no one has tested the effects of any change in technique or in techniques of interpretation on the ratio of correct to incorrect predictions. Therefore, although we cannot dismiss as meaningless the occurrences of correct predictions, we must be cautious about basing any claims on sporadic successes. These considerations underscore still further the importance of rigorous statistical analyses of analytic methodology and analytic validation.

In order to avoid any misconceptions it should be understood that an interpretation is not a prophecy. It is an hypothesis about the meanings of past events and about the confluent forces which have produced some pattern of behavior, thought and feeling. There are latent predictive implications in any interpretation; but these are not expressed. This implication is that if the hypothesis is correct, and if any one of the forces which constitute the described constellation should change, then the behavior will change. But the analyst does not make any such prophecy to the patient. At most the analyst may ruminate about these future events either silently to himself, or under certain special circumstances to a student, but not in the hearing of the patient and not to the patient.

If such implicit predictions were made to the analysand, then the fact that the analyst had ventured to make the prediction would introduce into the stream of the analytic process an element which would surely exert an influence on the subsequent chain of events, quite apart from the influence of the interpretation itself. Such a prediction would constitute what is called a self-fulfilling prophecy. But since the prediction is never made to the patient the self-fulfilling prophesy is not a problem which complicates this picture, or which must be considered here.

(7). It is almost impossible to speak about dynamic processes without using quantitative figures of speech, *i.e.* some kind of "more" and "less." Yet our right to assume that quantitative differences actually exist is not unlimited (17, 33, 42). Furthermore it is wholly invalid to assume that quantitative changes or differences are the only variables that can account for differences in activity,

in personality, and in illness. Today this whole area is being subjected to critical scrutiny by a number of analysts and critics who are concerned with the changes in thinking which the development of cybernetics, of information theory, of communications engineering, and of electronic computers, etc., are forcing upon us. Relevant here is the work, among others, of Pumpian-Mindlin (42), Warren McCulloch (34), Colby (7), Ruff (43), J. G. Miller (36–39), Ralph Gerard (12), Ashby (1–3), Grey-Walter (13) and J. B. Chassan (6).

(8). Two other grave deficiencies in our current conceptual equipment are closely related to this same problem of quantitative differences. These are 1) the lack of basic criteria of change (25, 27) and 2) the tendency of all systems of psychology to develop the illusion that a new description (which may be a more precise description or merely a new nomenclature for an old description) has greater *explanatory* value than the old. The distinction between description and explanation is of critical importance; yet theoretical formulae in psychoanalysis are usually hybrid and confused mixtures of the two.

(9). This leads to a consideration of certain instrumental lacks. I have already mentioned one, namely, that which derives from the fact that we depend predominantly upon the ear. This, however, is a lack which is being overcome. The use of infra-red motion photography will carry us forward another step by making it possible to study activity in the dark as in the light. Yet we still lack techniques by which to validate assumptions about specific quantitative changes. Most specifically we lack instruments with which to assay the relative roles which are played concurrently by conscious, preconscious and unconscious processes, and the changes in their roles which may occur during the course of an analysis. Paradoxical though it may seem, it is easier for us to study the impact of unconscious processes on conscious and preconscious activity than the impact of conscious communications on what goes on behind the iron curtain in the system of unconscious processes (14). Here again we need new instruments.

(10). As a result of these procedural deficiencies, we find ourselves with certain conceptual limitations. We are not yet clear as to (a) what constitute the internally cohesive clinical syndromes which have a common history and a common fate; or (b) the conglomerate drives which operate as conflicting and/or cooperating units in the personality; or (c) the basic personality units whose variations must first be described and then explained. These have been formulated and reformulated in many ways; and from time to time analysts have agreed about them briefly but never for long. Dissatisfaction with each formulation leads to fresh formulations, about which new disagreements occur. This is not unnatural; but it means that we have not yet completed the primary descriptive steps which are essential in the development of any natural science and which must be accurately done and tested before etiological hypotheses can be seriously considered. We cannot set out to explain something before we can agree on a description of the nature of what it is we are attempting to explain. (d) Nor is it clear how best to formulate the nature of the energetic processes, their original sources, the derivative forms which such energetic processes can assume, or the role which hypothetical differences in energy may play in psychological affairs. We have not yet been able to eliminate from our thinking the figurative language of the physics of the '80s and '90s; the physics of mechanics and hydraulic engineering, or the concept of electricity as a weightless fluid flowing through solid tubes. These images still dominate both analytic speech and theoretical conceptions. Thus in many directions a process of conceptual clarification is only starting. Only a few psychoanalysts have given up the primitive concepts that the activity of the central nervous system re-

quires the transfer of matter, or the transfer, increase or decrease of energy, or the transmutation of one form of energy into other forms, to replace these with concepts of the transmission of bits of information as a more appropriate conceptual model. A full discussion of these issues would necessitate a recapitulation and a thorough re-examination of the theory of instincts, of instinct derivatives and of their transformations under the impact of symbolic processes in man (25).[3]

Having listed some analytic deficiencies, let me turn to what I consider the essential virtues of psychoanalysis as an exploratory process, and why in spite of its glaring deficiencies I consider analysis to be of basic importance to all of science.

PSYCHOANALYSIS AS A SCIENTIFIC PROCESS

It is possible to divide psychoanalysis into components in many ways. Those which I will use have no special virtue, except insofar as they are helpful for the consideration of our problem.

The ultimate goal of analysis includes changes in basic components of personality, *e.g.* changes in the definition of personality boundaries, changes in identifications, changes in all conscience functions, reduction and realignment of areas of conflict, the reconciliation of irreconcilable goals, and the like. In its effort to move towards such long-run objectives the immediate and initial goal of analytic exploration is to illuminate the concurrent roles of conscious, preconscious and unconscious processes in human thought, feeling, purpose, behavior, and relationships. As a therapy psychoanalysis attempts to redistribute their roles, and to shrink the areas in which unconscious processes have a controlling influence. All of this, however, is outside the range of this paper, which is concerned with psychoanalysis for exploratory purposes. Therefore I will consider only the following elements of psychoanalysis as a process (27):

The constancy of the setting;
The constancy of attitudes;
The analytic incognito;
The role of interpretations as working hypotheses;
The role of transference and countertransference in interpretations and identifications;
The crucial significance of free association.

In the struggle to explore the inner space of human psychology and human personality, the basic challenge is the clarification of the respective roles of three "levels" or systems of concurrent processes, the interplay among them, and their individual and/or summated effects on behavior, thought, purpose and feeling in human life. I will say at once that there is abundant evidence, largely overlooked, that among these processes the most pervasive and continuous is neither conscious nor unconscious, but what we call preconscious. This seems to consist of a continuous and remarkably rapid stream of afferent, integrative, creative and efferent human mentation, mediated by coded cues and signals, but without fully developed symbolic representation: *i.e.* what the Wurzburg School used to call "Imageless Thought." In the waking state through the use of symbols our *conscious* processes pro-

[3] Freud's "economic" principle made the explicit assumption that it is impossible to explain either normal or abnormal psychology without assuming that they are based on differences in quantities of energy. This point of view reflected the concepts of the times, when physics thought only in terms of transmission of mass or of energy and the various transmutations of one form of energy into another. Even the transmutations of energy into mass and *vice versa*, which is a commonplace today, was only dimly perceived then. Today we are far more concerned with the transmission and processing of bits of information in the nervous system which occurs and varies with minimal investments or variations in energy consumption or output, just as it occurs in electronic computors. This simple fact is forcing a careful if somewhat reluctant revision of a great deal of psychoanalytic theory, terminology and concept. Miller (38, 39) points out that it gives us actually a new fourth dimension; *i.e.* "density," the number of bits of information transmitted and processed per unit time.

vide a relatively slow, limited and weighted sample of this continuous preconscious stream. Our *unconscious* processes constitute in turn an even more fragmented and distorted symbolic sample of the conscious samples of the underlying preconscious stream.[4]

In sleep preconscious processing constitutes a variable but apparently continuous preconscious dream-flow. Of this "The Dream," as we ordinarily know it, is only a condensed fragment, a weighted sample expressed predominantly through visual images. The result is a cluster of visual hieroglyphics with multiple simultaneous conscious, preconscious, and unconscious implications. No matter how we structure the personality or organize it, no matter how we use such conceptual metaphors as id, ego, and superego, or regression, defense, identification, incorporation, denial, reversal, etc., since each and every one of them operates on all three of these levels concurrently, the critical problem remains the same: *i.e.* can we determine the relative roles of these three concurrent systems of processing and sampling, and their interplay? To determine this will require the development of techniques by which to isolate the influence of each of the three systems of mental activities as they operate concurrently in human affairs. At present, faulty though it is, psychoanalysis is the only instrument which even approximates this goal. That out of it more precise instruments will evolve I do not question. But these will be rooted in the pioneer work of Freud.

I have stated this position without indicating in detail wherein this thesis is an extrapolation from the position which Freud took many years ago, and which he then allowed to fall virtually into disuse but without ever disowning it. His scientific imagination seemed merely to be drawn in another structural direction. One might think of this period as a tide which drew him to an anatomical rather than a physiological shore, paralleling perhaps his earlier neuroanatomical interests. Because of this shift in emphasis in Freud's theoretical structure, the description given here is an extrapolation from Freud's earlier position, but varies in certain important ways from his later "structural" metaphors and from those current modes of psychoanalytic theory which derive from his structural concepts. This seems to me to be one of the virtues of my point of view, because the structural metaphors seem to me to have long since proved their sterility. But the reader should be warned that there are other experienced and thoughtful analysts whose thinking conforms more closely to Freud's later schema and who do not agree with me.

What troubles me far more is the difficulty of giving adequate operational descriptions of how to differentiate among conscious, preconscious, and unconscious processes. It is easy to characterize their consequences clearly. This I have done in many previous articles, pointing out the easily recognizable characteristics of behavior in which conscious processes dominate the field of forces (24); and also the characteristics of behavior in which preconscious processing flows freely, dominating the psychic stream and furnishing a continuous supply of processed experience for symbolic sampling (30, 31). Nor has it been difficult to describe the special qualities of behavior which is dominated by processes in which the link between the conscious symbols and their unconscious roots has been severed or distorted, which is what occurs in unconscious processing. Unhappily, however, to separate out three types or levels or systems of psychological process through their characteristic effects on behavior is not the same as recognizing, isolating, characterizing and ultimately measuring the role which each is playing in a stream of events and processes in which the three are intermingled. We can state with

[4] Note that in the Unconscious System it is never the symbol or the symbolic act which is unconscious, but its relation to that which it is supposed to represent, but masks instead.

considerable confidence that human experience can be perceived, processed, represented, (and also misrepresented) and responded to in three ways. We can recognize these three ways from their results. But this is an indirect process of identification. An operational definition in terms of the techniques by which each can be isolated and measured is not yet within our grasp. For years I have urged our friends among the clinical psychologists and neurophysiologists and electroencephalographers to set themselves the task of devising test instruments which will provide methods by which to sharpen our formulations of these three concurrent methods of dealing with experience, tests which might appraise the relative role of each in any one moment of behavior, and which might even give us an indication of those areas in life in which conscious and preconscious processing predominates as opposed to those areas in which unconscious processing holds the upper hand. This differentiation is rendered all the more complex by a fact which has been emphasized already, namely that none of these processes ever operates alone, all three always operating concurrently.

This then is a basic challenge to us. We recognize its importance fully; but we cannot yet cope with it adequately.

Let me summarize by indicating again that I characterize the primary exploratory goal of the analytic process as an attempt to clarify the continuous interplay of concurrent conscious, preconscious and unconscious processes in every moment of life, whether awake or asleep, and in every aspect of human personality and behavior. Their interaction enters into every creative act in science, art, music or literature, and into all neurotic and psychotic processes as well, with or without organic substrate. Therefore this interaction is at the core of our understanding of all human psychology, sick or well. Implicit here is a concept of the relationship between neurosis and health which involves the essential nature of the neurotic process, its genesis, and the role of conflicts on all three levels. It is not my purpose to discuss this concept here. Instead, I will refer to previous studies of the question (24, 30). But with this premise in mind, we can now consider in what analysis *as a process* consists:

(1). The framework of the analytic interview maintains to a unique degree a relative constancy of external variables, making it possible to repeat observations and to collect data day after day under relatively unchanged external circumstances. The situation is sufficiently standardized to enable us to make comparable observations on one person at different times, and also on different people.

(2). The formality of this framework cannot eliminate the fluctuating state of the *observer* (to wit, the analyst); but it limits the impact of these fluctuations on the interaction between analyst and analysand and therefore, on the process of observation. Thus the analyst's "incognito," his formal aloofness, his avoidance of any extra-analytic role or contact, all help to minimize and to render constant the distortions which would otherwise be interposed by the analyst's unconscious processes, helping him to approximate the role of a relatively passive and inert observer and recorder of his own percepts of the patient's productions.

(3). A situation results in which the majority of the spontaneous variables which are introduced into the stream of material arise out of the subject of the investigation—the analysand. This makes it approximately possible to study the mind of another without distortion and interference from that of the observer.

(4). The analyst makes his own conscious samples and conscious summaries of his conscious and preconscious responses to the subject's productions; and from time to time he shares these with the patient in the form of "interpretations," (the term the analyst uses for his working hypotheses.)

This requires further explanation. By re-

maining as ill-defined as is humanly possible, by masking his own emotional reactions, his esthetic tastes, his political and religious views, his family ties, the details of his personal life, and his feelings about the patient and the patient's communications, the analyst attempts to remain a cipher about whom the patient can imagine anything he feels and feel anything he must. The analyst's goal thus is to be a blank human screen, on whom the patient can project his concurrent conscious, preconscious and unconscious feelings and fantasies. As long as the analyst as a real person remains essentially unknown to the patient, the patient's thoughts, feelings and fantasies about the analyst will not be earth-bound to reality. Instead they will be products of the patient's imaginative and creative psychological potentials. To the extent to which the analyst remains unknown, he will be a sample of humanity drawn from a grab-bag; and on this relatively blank image of the analyst the patient can then drape his fantasies, as one might drape a costume on a dummy in a store window (19).

The study of this process in the analysis gives the patient insight into the hidden and subtle ways in which similar projections can distort any and all of his daily human relationships; (*i.e.* his response to the stranger he passes on the street, to the acquaintance who greets him, to a boss, to an associate, to a child). Gradually the fact becomes inescapable to him that these are all projections of his own unconscious needs, conflicts and fantasies, many of them transplanted from the remote past. In short, the analytic incognito makes it possible for the patient to use the analyst as though he were an animated, mobile, more-or-less symmetrical stimulus card in a Rorschach or a Thematic Apperception Test, *i.e.* an animate object on which to project his feelings and fantasies. The study of the distortions of reality which such projections produce illuminates for the subject the distortions which can enter into all of his personal relationships. This is part of what is meant by the study and clarification and analysis of transference processes.

Conversely, if the analyst allows himself to become known to the patient, he will perforce anchor the patient to reality, thus obscuring the role of the patient's unconscious needs and feelings.

Note that I have not said that any of this *eliminates* counter-transference processes. We are justified only in saying that the conscientious maintenance of the analytic incognito lessens the impact of counter-transfer ence processes on all three levels, and reduces significantly the distortions which they can otherwise introduce. These considerations have been more fully explained in other available studies (19).

The question is often raised whether the analytic incognito is a fiction or a fact. Is it not inevitable that even if the analyst is unknown at the start of the analysis, he must lose some part of this incognito each day, each time he greets a patient or says good-bye, each time he speaks, each time he offers an interpretation? Over the course of an analysis does not the analyst's incognito suffer gradual decrements through the cumulative effects of minute cues, both subliminal and conscious? There are the daily differences in expression, posture, gesture, intonation, dress. Do not all of these betray to the analysand much about the analyst as a person, his moods and his convictions, his allegiances and his vulnerabilities? Of course this is true. Unless he wore a mask and gown, or analyzed from behind a screen, and made all of his comments and interpretations by typewritten notes, or had them transmitted in some stereotyped fashion and in a voice not his own, the patient must gradually accumulate clues about him. Yet such clues are faint and ambiguous; and into the analysand's interpretation of them he will project his own fantasies. Consequently, as long as the analyst keeps his distance and holds in check his own exhibitionistic impulses, the analytic value of the patient's

fantasies about him will not be lost merely because the patient gradually picks up uncertain clues as to the real nature of the man behind the analytic mask.

For certain investigations it might well be important to eliminate even such faint clues as these. The analyst's presence might be represented by something as impersonal as a typed card, or by a voice electronically altered in transmission so that its modulations would be obscured. For ordinary purposes one stops short of experimental perfection, and in therapeutic or training analyses the analytic incognito is relative and not absolute.

Nevertheless the more intact is the incognito, the greater is its value for translating transference forces and also for evaluating the validity and the impact of any interpretation. This is because when an interpretation is made from behind the screen of an intact analytic incognito it constitutes a single variable introduced into a *relatively* constant analytic interaction. Without the incognito, the same interpretation would drop into a turbulent sea of complex waves of transference and counter-transference, in which so many variables would be interacting that the interpretation of the role of any one of them would be almost impossible. Consequently the effort to maintain as strict an incognito as is possible conforms to an elementary principle of scientific research, namely, to aim always to introduce one variable at a time.

If the psychoanalyst, furthermore, is scrupulous about not establishing any extra-analytic contacts with his patients, the analyst will avoid an array of additional sources of dangerous confusion. Once an analyst establishes a social relationship with his patient, he begins to make demands on the relationship which serve his needs rather than the patient's. Thereafter the analyst's unconscious conflicts will play an increasing role among the processes which shape his interpretations. Self-serving purposes become confusingly interwoven with his efforts to be patient-serving. Therefore both for scientific and for therapeutic purposes it is essential for the analyst to eschew extra-analytic contacts with his patients. There can be no compromise about this principle.

In small communities the fact that everyone knows everyone else creates a serious obstacle to the analytic incognito. In such centers there are only a few analysts and the people who seek to be analyzed, whether for educational, cultural or therapeutic purposes, inevitably come from a small in-group. Everyone who has worked in small university towns or in research institutes or psychiatric hospitals which are remote from large centers, knows how difficult this situation is and how it tends to contaminate the analytic process. Here as in so many activities in life, practical exigencies force realistic compromises; but this does not alter the scientific principle. A surgeon who is operating in a jungle may have to operate without benefit of sterilization. He may have to accept the risks involved and do the best he can. This does not change the fact that surgery in a sterile field with clean hands and clean instruments is better surgery than surgery with unsterile hands in a dirty field. Similarly analysts who work under unfavorable circumstances may have to make comparable compromises. This does not alter the fact that for the scientific investigation of the analytic process itself and in the use of analysis for psychological exploration, the incognito is essential. Although this control is not our concern here, there is every reason to emphasize its importance in therapy as well. Therefore to conduct an analysis in an uncontaminated field, *i.e.* under circumstances in which the analytic incognito will not be continually invaded and penetrated, is essential for its scientific use and exploration.

Finally, I would emphasize the fact that transference and counter-transference are not peculiar to psychoanalysis. They operate in every human relationship. But they are studied in analysis as in no other procedure.

At this point I must remind the reader that I am not considering therapeutic leverage, but only the scientific and exploratory value first of something which is called the analytic incognito and second, of the analysis of transference and counter-transference processes. These devices are essential for protecting the material which the analyst is observing from distortions through contamination by the observer.

FREE ASSOCIATIONS AND THE ANALYSIS OF TRANSFERENCE PROCESSES

SIGNIFICANCE OF FREE ASSOCIATIONS

It is strange that the challenge of free associations has been so neglected even by analysts. This failure is evidenced by the absence of critical investigations into variations in the patterns of free associations in response to variations in neurophysiological and psychophysiological conditions. This lack has in turn been one of the major obstacles to the steady growth of psychoanalysis as a science. It is fair to add, however, that such studies have become possible only since automatic recording has been readily available. I have already indicated that it is impossible to listen to ordinary speech without distorting our perceptual records of it. It is even less possible to take in and record and recall a string of free associations. They are distorted first at the time of perception, and again to an even greater degree in retrospect: this is true no matter how phonographic the mind of the listener. We can remember an entire sentence as a unit, but if we break up that sentence into syllables and scramble them, it becomes impossible to remember the same number of "nonsense" syllables. Yet free associations, when they are truly free in the technical analytic sense, approximate strings of ideas and words or even fragments of words and ideas without apparent relevance or pattern. Consequently, the task of perceiving and recalling truly free associations becomes so difficult that only a recording device can provide the raw data of analytic observations for critical qualitative and quantitative analysis as to content, meaning and form.

Such free associations are important for many reasons. First, they constitute a mirror image of the conditioned reflex, as was first pointed out in 1934. Their deeper importance arises out of the fact that this is our only approximation to a technique by which to secure relatively unweighted and therefore representative samples of *all* that is going on in the mind at any one time. Any conversation which is goal-directed, any effort to talk about a chosen topic, involves an automatic, preconscious process of selection and rejection. When I am talking about baseball, I cannot at the same time talk about football; and if I talk about football, I cannot talk about science or philosophy. From the conscious election of a topic to talk about flows a preconscious process of screening, selecting and rejecting. This goes on all the time at extraordinary but still unmeasured and uncalculated speeds. Yet it is what makes human speech possible. Under these circumstances the spoken word is only a selected and weighted fragment of all that is going on in the speaker's head at any moment. Clearly then when we need a more representative cross-section of another's mental process, we must conduct an approximation of a Gallup poll. This we can do only if the speaker talks without electing to focus on any topic, or to exclude anything which comes to his mind, so providing the raw material of free associations. By not excluding or selecting anything *deliberately*, free associations become free of *conscious* choice, selection or rejection; and by the same token, automatic preconscious selection and rejection are at least minimized. What is unmasked thereby is the influence of *unconscious* processes on the preconscious linkages among feelings, ideas, actions and purposes, which are themselves the products of conditioning (16, 30). It is this which gives the technique of free association its unique significance as a method for unmask-

ing the concurrent influence of conscious, preconscious and unconscious processes in mental activity. Glover said long ago that as long as free associations are flowing, an analysis is moving.

DEPENDENCE OF FREE ASSOCIATION ON THE ANALYSIS OF TRANSFERENCE PROCESSES

We have seen how the analytic incognito assists in the clarification of these special projections which constitute transference processes. The next step is to see how this in turn helps the patient to use the critical exploratory tool of analysis, the technique of free association.

If free associations are to be studied they must be communicated. It is therefore essential to minimize all factors which impede their *communication*. Among such factors, unacknowledged and unconscious transference attitudes play a dominant role, and their resolution through interpretation is therefore of vital importance.

Up to the present time no experimental or observational situation has been designed which for this specific purpose is comparable to the analytic situation. Every element in formal analysis contributes to the eliciting of free associations; to wit, the analytic incognito, the constancy of external variables, the non-interference by the analyst, the interpretation which is introduced as a deliberate and variable working hypothesis, the watchful eye on transference and counter-transference pressures and the tracing of such attitudes to their sources. All of these are essential for the facilitation both of the production and of the communication of free associations. They do not facilitate the easier gratifications of conventional conversational interchange. On the contrary, they rigorously reduce the opportunity for such gratifications.

This is not an over-simplification: it is the core of the matter. Although as stated it sounds easy, in practice it is difficult and elusive. Few individuals, whether as patients or as participants in experiments, can produce free associations in this precise sense for more than a few intermittent moments, scattered among hours of automatic defense against it. The potentially facilitating influence of drugs or of other physiological variables have not yet been adequately explored, either in qualitative or quantitative terms.

We should challenge ourselves next with the question, "How do we know that associations are *free*?" If we are honest, we must reply that we do not yet *know* in any ultimate sense; that of this we have only approximate knowledge; and that we will not have conclusive indications of how truly free free associations are, until through the application of recording methods and their meticulous quantitative and qualitative study (including timing) we secure more precise data than has yet been available as to the nature of such material. When this type of research is carried out, we might find that there is discontinuity or a continuous spectrum or a spectrum with bands of clustered characteristics stretching from the bound associations of goal-determined speech to the freer aspects of the products of free associations.

Furthermore, in all such investigations the rigorous application of the techniques and principles of the developing discipline of linguistics and para-linguistics may play an important contributory role.

FURTHER AREAS FOR RESEARCH

(1). Implicit in the formulation discussed above are the differences between the coded signals which mediate preconscious processing, the symbolic process which mediates conscious sampling, and the distorted symbolic process of "unconscious" sampling. A full discussion of these issues would take us far afield. Here I would emphasize only the fact that the symbolic process has vital implications for the whole concept of the human apparatus as a machine.

(2). It is through the interposition of coded signals and symbols that the human system

can anticipate any thrusting needs from within or any thrusting impulse from without; and because of these anticipatory warnings it becomes possible for the human machine to make needed corrections *before* the responsive deviation occurs. This indeed is the essential distinction between instinctual processes in the human animal and in other animal forms, insofar as they have been studied (25). And again this is an area of psychoanalytic thought which must be reformulated by analysts in their search for clarification of the analytic concept of instincts, as a basis for inter-disciplinary work in this field.

(3). Analysts must also turn their attention to the role of all preconscious activities; *e.g.* the incessant bombardment by subliminal stimuli in all afferent modalities, internal and external; the sub-symbolic processing which occurs within the human machine to implement all preconscious activity and which corresponds to the coded signal of the communications engineer. These processes and their variations from one man to another and in different states of illness have not yet been made the object of specific study.

(4). This leads directly to still another area for basic research, which derives from the newer knowledge of feedback and cybernetics and their relationship to psychological functions in general and to psychoanalysis in particular. A tendency is inherent in all feedback mechanisms to minimize any stimulus-response deviation. This is recognizable in all central nervous system activity from the reciprocal innervation of Sherrington to the Freudian concept that pleasure is characterized by a reduction of tension. For this reason the verbal model which psychoanalysis derived from the pre-existing concepts of physics is wholly inappropriate. Consequently not only our language but our thinking about the dynamics of change in mental phenomena must be wholly recast. Without going further into these questions, it may be predicted that in the long run the application of principles based on models derived from modern electronic engineering, information theory, communications engineering, mathematical machinery, digital and analogic computers and electronic devices in general, will clarify much that at present is confused in psychoanalytic theory, concepts and terminology, and eventually will furnish analysis with further techniques for critical self-examination. Instead of transfers of material or of energy or even its transmutation, analysts will find themselves concerned with the reception, storage, processing, transmission and reproduction of individual or clustered bits of information.

THE PHILOSOPHIC POSITION

It is necessary, finally, to discuss briefly the philosophy that is implicit in the analytic assumption that dynamic interactions can occur among psychological events. This issue was spelled out more fully in a paper which was written several years ago, but which appeared relatively recently (29), entitled "The Psychodynamic Position on Etiology." There is much in the body of that paper that I would change if I were writing it today; but with only a few essential changes for the sake of clarity and accuracy, the philosophical statement stands up fairly well, and will be condensed here.

Any consideration of dynamic interactions on the level of psychological experience stirs a venerable philosophical conundrum out of its Rip Van Winkle slumbers. Fortunately, although the problem is ancient, I have the temerity to believe that it can be resolved simply. The idea that sequences of psychological events can exert dynamic influences on one another seems at first thought to do violence to the scientific spirit, and to the laws governing the conservation of mass and energy. Yet this need not worry us. There is no difficulty in understanding in physical terms how perceptual stimuli which arise from *immediate* occurrences initiate sequences in the central nervous system, both chain reactions and reverberating circuits, and thereby influence the behavioral and

symbolic (*i.e.* psychological) expressions of central activity. Such units of input initiate coded signals which transmit further processes and give rise to new chains of coded signals, and finally to their symbolic sampling.

Furthermore, because of the low level of energy interchange in reverberating circuits within the nervous system, once these processes are launched they can continue in transmutable forms even for years. In this sense they are "stored." Thereafter, any symbol which represents past or spatially distant events, or events which are anticipated in that abstraction which we call "The Future," can serve as new coded signals for some nervous apparatus in the here and now; and those new coded signals in turn trigger off or redirect or redistribute further processes. We are, therefore, merely using verbal shorthand when we talk of cause and effect on the psychological level. To say that one state of mind causes another, whether in me or in someone else, is a legitimate semantic elision. We do not need each time to go through the cumbersome and pedestrian steps of saying that in my mind preconscious coded signals and/or the conscious symbols of one moment touch off a chain of preconscious coded signals and/or conscious symbols, either in my own mind again or in the mind of someone else to whom I have communicated these symbolic samples by word or gesture. The shorter statement does no violence to our basic concepts of the physical world, provided that in our theoretical constructions and in our experimental work we do not forget that in this process, within one person, the effective stimuli to each successive step are the coded signals for internal communications, whereas from one person to another the effective stimuli are the symbolic cues which are communicated from man to man.

From this we may generalize further to say that it is the preconscious coded signal or its conscious symbolic counterpart which enables one psychological state to influence another. It may seem strange that we even stop to explain this, since its occurrence is an elementary fact of human experience. If it did not happen our past could never influence our present or future, nor could there be any communication on the level of psychological interaction. No mere event could then have a lasting influence on anyone. Yet we know that the thoughts and feelings of today are integral with those of tomorrow; and that words, expressions, gestures and events "communicate with one another," in this manner of speaking. We also know that my symbolic activities, acting through perceivable symbolic or subliminal signals, are incorporated into the psychological processes and ultimately into the personalities of other human beings, whose symbolic and preconscious responses in turn and in the same way become incorporated into mine. In short, in all goal-directed animal behavior, superimposed upon the preconscious devices for coded signalling within the machine, are the devices for symbolic sampling, processing and communicating. Comparable sequences are thereby initiated within each of us.

Moreover, once my own symbolic cues have been activated, they become events to me which are quite as potent as are the symbolic attitudes, expressions, gestures, and acts of a friend or stranger. It is this which stamps the human race with its uniqueness, and the extraordinary development of which distinguishes man from even the highest primate. *Preconscious coded signalling* is the instrument by which experience is processed within us on the psychological level and can be translated into somatic changes, or conversely somatic experience into psychological change. *The symbolic process* is the instrument for self-criticism and rumination and for deliberately communicating signals, information, and interactions. Together these are the instruments of our greatest creative power, but also of our greatest vulnerability (24, 30). Furthermore, although both systems are vulnerable, of

these two (*i.e.* the mechanisms of conscious symbolic sampling and of preconscious coded signals), the conscious symbolic process is patently the more vulnerable to distortions which are of organic or psychogenic origin.

Evidently then the coded signal and the symbol are the two psycho-physiological implements, an understanding of which is essential for an explanation both of normal and of pathological human psychological function. This is the key concept in psychodynamics and psychogenesis, whether of normal or sick activity. That from earliest childhood the symbolic process can go off its rails is perhaps the greatest challenge that medicine faces today; because if this did not happen, there could be neither the neurosis nor the psychosis as we know it in the human being.[5]

At the same time the symbolic representation of remote events binds widely separated times and far places into one immediate interacting psychological continuum. This is one of its several essential functions; and whereas on conscious and also on preconscious levels we can sharply distinguish the past from the present and the near from the far, on the obscure levels of dynamic unconscious processes, near and far, past, present and future fuse into a continuum so complete that differentiation among them is lost. This process was first described clinically by Freud (11). More recently it was demonstrated experimentally by Penfield (23, 41).

We can summarize this brief salute to an ancient philosophical dilemma by stating that in the study of dynamic interrelations on the level of psychological experience, no assumptions which violate basic scientific concepts of the world are needed; since by means of symbols (whether these are conscious or whether they function as preconscious coded signals) effective and sustained interactions can occur among sequences of psychological experiences. The symbol turns abstraction into a concrete stimulus.

[5] This is far from being all there is to the psychopathological process; but this is the aspect of it which is most relevant to our philosophical dilemma.

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CHARACTERISTICS OF DEPRESSIVE AND PARANOID SCHIZOPHRENIC REACTIONS ON THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY

W. GRANT DAHLSTROM, PH.D.[1] AND ARTHUR J. PRANGE, JR., M.D.[2]

Although it is now twenty years since its development and fifteen years since it was generally distributed, the Minnesota Multiphasic Personality Inventory (MMPI) (11) is still the only empirically derived, objective personality instrument available in the field of psychiatry. A great deal of work has gone into the development of its basic clinical scales, its validity scales, and the nearly two hundred additional measures derived from this item pool (25). There is still a need for more detailed analysis of MMPI results on relatively homogeneous diagnostic groups, particularly the various score patterns that are related to specific symptom patterns.

The basic variables in the clinical profile of the MMPI include: 1. Hypochondriasis (Hs); 2. Depression (D); 3. Hysteria (conversion) (Hy); 4. Psychopathic deviate (Pd); 5. Masculinity-femininity (Mf); 6. Paranoia (Pa); 7. Psychasthenia (Pt); 8. Schizophrenia (Sc); 9. Hypomania (Ma); O. Social introversion (Si). In addition there are a Cannot Say (?) score, a Lie (L) scale, a validity (F) scale and a defensiveness correction (K) scale. The clinical scales were constructed singly against specially selected diagnostic groups by contrasting the test responses of each of the groups with a large group of normal adults. The items indicative of each group were gathered into separate scales. The whole set of scales now is used to form a psychograph or profile. Each patient's test is scored for all of the scales and the profile provides a set of comparisons between the individual and each of the criterion groups.

Although the early work on the test was directed to the component scales, it soon became clear that most patients showed a considerable degree of similarity to more than one psychiatric group. That is, any one patient was unlikely to rely upon a single set of defenses, but would show at any one time many different defensive mechanisms. Unfortunately, it is not now known how the patients that made up the criterion groups used in building each of the original scales reacted to the rest of the scales in the test. In addition to the basic similarity that led to their being members of one of the criterion groups it is quite likely that the patients in any one group would have shown important differences, one from another, in MMPI test pattern. Few studies have since reported on pattern frequencies within homogeneous subgroups of psychiatric patients. Those that have appeared, like Fricke's (5) on 63 females with conversion hysteria, have typically not provided a breakdown on the profile types appearing or of the relative frequencies of each pattern. The purpose of this study is to show the patterns associated with two major psychiatric reactions and the role they play in the behavioral manifestations of these psychotic conditions.

DEPRESSION

The derivational work on the scale for depression (D), scale 2 (10), was based on a carefully selected group of cases suffering from relatively uncomplicated depressions. Nevertheless, the test authors found it necessary to add some items to the scale

[1] Department of Psychology, University of North Carolina, Chapel Hill, North Carolina. The authors wish to acknowledge their indebtedness to Harley C. Shands, M.D. for emphasizing the need to refine the distinctions between and among depressed and paranoid patients.

[2] Department of Psychiatry, University of North Carolina School of Medicine, Chapel Hill, North Carolina.

to increase the specificity of elevations on this scale. Many psychiatrically disturbed patients not suffering from prominent degrees of clinical depression would otherwise have earned high scores on scale 2. These additional correction items helped to reduce this spurious kind of D score. It should be obvious, however, that any valid measure of depression is going to be significantly elevated in patients presenting any of a wide variety of personality reactions. This would be so, if only because their defenses are failing to keep these patients sufficiently adjusted to stay out of medical treatment. Although there has recently been some work on the D scale itself (3, 5, 17), the scale now used in clinical practice is the same set of items chosen in the original derivational work. Most of the work since this time in the evaluation of clinical depression has been based on profile patterns.

The available data in the literature on these depressive groups are summarized in Figure 1 from the studies of Leverenz (14), Pacella *et al.* (18), Guthrie (7) and Rosen (19). Some of these reports give only incomplete data on the score components, particularly the validity scale values. These studies also vary in the care of patient selection and in the proximity of the test administration to the time of the diagnostic appraisal, a point which is of special significance in such a transitory state as depression. The group profiles in these four studies show a great deal in common: the peak score is on the D scale; the general elevation of the profiles is quite similar; scale 7 (Pt) is either the second highest scale or is tied for second place; scale 9 (Ma), when reported, is the lowest scale in the profile; among the first three scales in the profile, Hs, D, and Hy (the neurotic triad), the D scale exceeds the others by at least ten T-score points, while Hs and Hy are usually within a point

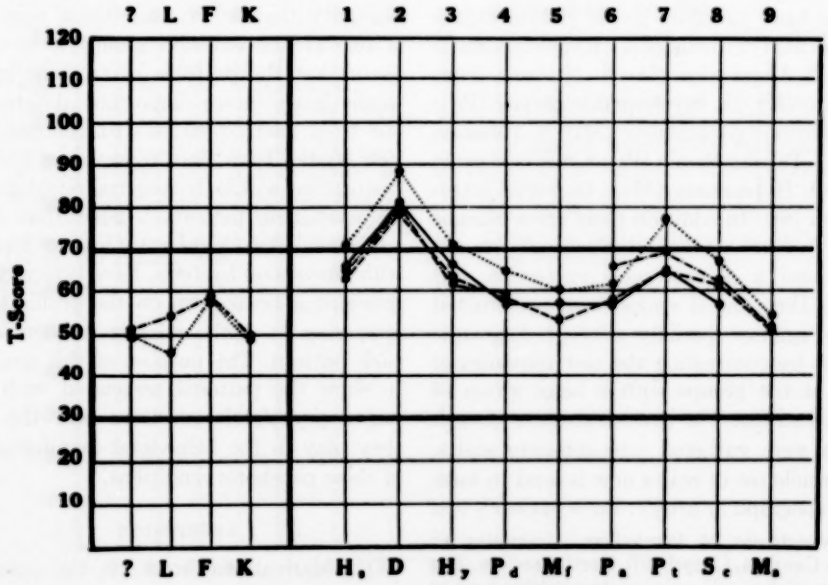

FIG. 1. MMPI profiles from various depressive groups.
—— Leverenz (14). Code: 2″ ′137-4568. (N = 28)
– – – Pacella et al. (18). Code: 2′7138-4659. (N = 24)
-·-·- Guthrie (7). Code: 2″ ′76138-49 FL?/K. (N = 7)
···· Rosen (19). Code: 2″713′84605-9 FK?/L. (N = 36)

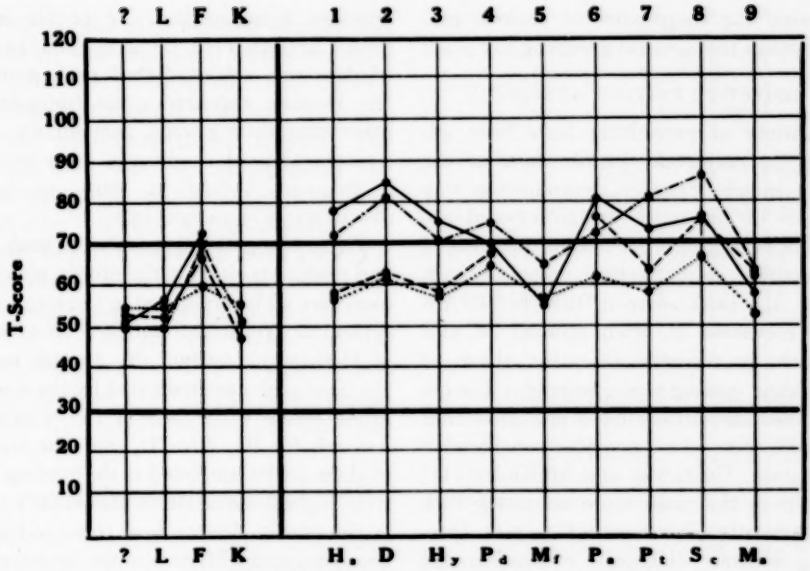

FIG. 2. MMPI profiles from various paranoid groups.
—— Gough (6). Code: 26″1837′4-95 F′L?. (N = 11)
– – – Guthrie (7). Code: 68/4792-13 F-?L/K. (N = 15)
-·-·- Rosen (19). Code: 827″4613′59 F′-KL. (N = 100)
···· Wauck (22). Code: 8462-79351 FKL?/. (N = 31)

of one another. These averaged profiles conceal considerable variation, however, from one patient to another within these groups; these variations can be handled only by some sort of pattern analysis.

PARANOID DISORDERS

The scales for paranoia (Pa), scale 6, and for schizophrenia (Sc), scale 8, were also derived empirically against criterion groups (*Cf.* Hathaway in (9)). A great deal of work went into this original development and, in the case of scale 8, additional refinement of its diagnostic utility was carried out in the K corrections now routinely applied to this scale. Although some MMPI items were used in Aronson's (1) work on paranoia, only the means for scale 5 (Mf) were reported, no use being made of scales 6 or 8. He noted that while the VA patients with paranoid diagnoses were higher than normals on scale 5, they scored no more femininely than did a sample of other psychiatric patients. Profile data on various forms of paranoid disorders are shown in Figure 2 from the studies of Gough (6), Guthrie (7), Rosen (19) and Wauck (22). It is clear from this set of profiles that the paranoid groups show greater differences in group patterns from one to another than do the depressive disorders. For one thing, these groups were formed on quite different bases using less stringent case selection procedures. Although the Pa scale is prominent in these profiles, it does not have the same clear-cut role in this set that the D scale had in the previous set. The neurotic triad (scales 1, 2 and 3) varies widely in elevations from group to group, probably as a result of differences in the acuteness of the psychotic reaction being sampled in each group. There is an obvious need for further material to clarify these profile characteristics, as well as to

determine the frequencies of various patterns within this general grouping.

OBJECTIVE PATTERN ANALYSIS

A variety of procedures have been advanced to refine the profile data of the MMPI in an objective, reproducible way (4). One of the most straightforward approaches is to group profiles on the basis of the score that is relatively the most deviant, that is, the peak score in the profile. Absolute elevation is often ignored in this approach, or reported as either above or below some cutting score, usually a T score of 70. Several writers have indicated that this peak score alone provides considerable information. Hathaway and McKinley (11) report that the peak score coincides with the diagnostic placement of a case (presuming accurate diagnostic evaluation and an MMPI administered close in time to the diagnostic appraisal) in about half of the cases. The data listed above already suggest reasons for deviations from this relationship, since depression may even play an important part in cases diagnosed as having paranoid disorders. Karson and Freud (13) using cases from a factor study by Borko (2) indicated that assignment of cases into diagnostic category on the basis of peak score alone was the most accurate method in their study, in contrast to clinical interpretation of the profile configurations. The overall accuracy of this method, however, was not very high.

Modlin (16), although not reporting a composite profile of his depressive group, gave some preliminary patterning results. In his group of 31 depressed cases seen in an army hospital, 88 per cent had their peak score on the D scale. The other peaks occurred on either Hy or Sc. The relative elevation of a given scale is also pertinent in patterning studies. In 97 per cent of Modlin's group, the D scale was among the highest four scales in the profile, even though there was no close check on the time of administration of the MMPI during the depressive episodes being studied. No profile in this group appeared to be a normal pattern. Modlin also indicated that peak scores on the D scale appeared quite frequently in other diagnostic groups, particularly in the hypochondriacal conditions. This has been a frequent finding in acute psychiatric disturbances of various sorts.

These two patterning features, peak score and relative position in the profile, plus many more, are all incorporated in the total coding system of Welsh (23) which is an extension of Hathaway's system (8). In this method the basic scales are indicated by the numbers given above with each of the scales, *i.e.*, 1 stands for Hs, 2 for D, etc. The numbers of these scales are listed in descending order from highest elevation in the MMPI profile to the lowest. Examples of these codes have been provided with each of the figures above. Next, elevation signs are inserted to indicate different T-score levels: 90 and above, *; 80–89, ″; 70-79, ′; 60–69, —; 50–59, /; 40–49, :; 30–39, .; scores below 29 are not further separated. The last step involves indicating scores falling within one T-score point of each other by underlining them.

In this system, peak scores can be identified by the number of the scale starting the code. The place any one scale occupies in the profile can be determined by finding the appropriate number and noting the scales above or below it (or tied with it within one point). The general elevation of the profile, the scales that fall consistently low or high, as well as specific intra-profile features can be easily identified.

PROCEDURE

Records from a state hospital were examined to locate fifty valid MMPI profiles in each of two diagnostic groups: psychotic depressive reactions and paranoid schizophrenic reactions. Included in this group were a paranoid state, a paranoid type of involutional psychotic reaction, and one paranoia. The patients were white, ranged in age from twenty to fifty-nine, and were at

least dull normal in intellective competence. The testing was completed within ten days of the admission on which they had received the criterion diagnosis, but not all cases were first admissions to the hospital. The diagnosis used was the consensus of the clinical staff in a conference held on each patient after ten days of hospitalization.

In addition to the criteria listed above for inclusion in the diagnostic groups, additional requirements were imposed on the MMPI protocols. No record with more than 60 omitted items was included, nor was a record with an L score over 10 raw score points retained. No further restrictions were imposed.

EXPERIMENTAL SUBJECTS

It was not possible to equate these diagnostic groups for age, sex composition, or other background variables. The depression group is composed of seventeen males and thirty-three females, while the ratio in the paranoid group is fourteen to thirty-six. Table 1 summarizes the age level of each of the subgroups.

RESULTS

In Table 2 the means and standard deviations on each of the MMPI scales for each of the subgroups are listed. Figure 3 presents the mean profiles for the male and the female depression cases. Figure 4 shows the corresponding profiles for the paranoid subgroups. Tests of the significance of differences between group means on the MMPI variables indicated that in general the depressed and paranoid patients differed from one another at a stable level on all scales except 5 (Mf), 7 (Pt), and O (Si).

To allow comparison with Modlin's group, the high-point frequencies for each sex within each diagnostic group are listed in Table 3. Tables 4 and 5 provide a more complete breakdown of profile patterns found in each of the diagnostic groups in terms of two-point high-point combinations. Within each of the columns are listed by code number the percentage of cases that had a given clinical scale as the highest value in the profile and the percentage of cases with each of the other scales as second highest value.

TABLE 1

Means and Standard Deviations of Ages of Patients in Each Diagnostic Group

Value	Group			
	Depression		Paranoid Schizophrenia	
	Male	Female	Male	Female
N	17	33	14	36
Mean Age	50	45	35	37
Standard Deviation	6	10	7	10

TABLE 2

Means and Standard Deviations of the Basic MMPI Scales for Each of the Diagnostic Groups

MMPI Scale	Depression				Paranoid Schizophrenia			
	Males		Females		Males		Females	
	M	SD	M	SD	M	SD	M	SD
?	51	15	49	13	53	4	55	2
L	60	9	60	10	57	10	56	5
F	59	8	60	11	67	11	71	15
K	58	15	56	10	54	10	51	9
1 (Hs)	76	18	69	10	57	14	61	14
2 (D)	86	20	81	22	67	17	64	16
3 (Hy)	75	16	73	10	55	12	62	16
4 (Pd)	64	10	65	19	70	16	72	13
5 (Mf)	54	11	54	12	57	13	55	13
6 (Pa)	67	15	66	16	67	12	75	18
7 (Pt)	70	21	69	13	66	18	65	16
8 (Sc)	67	21	67	15	70	15	76	14
9 (Ma)	54	13	55	12	55	16	65	19
0 (Si)	59	10	63	10	59	11	59	11

Although no systematic attempt was made in this study to develop a pattern combination for differentiating these two diagnostic groups, when a cross-validational sample becomes available, the system of Sullivan and Welsh (21) or some equivalent will be applied to these patterns. However, there was an opportunity to test Strong's (20) Hy-Pd profile sign which he discovered in his analysis of extremely elevated records

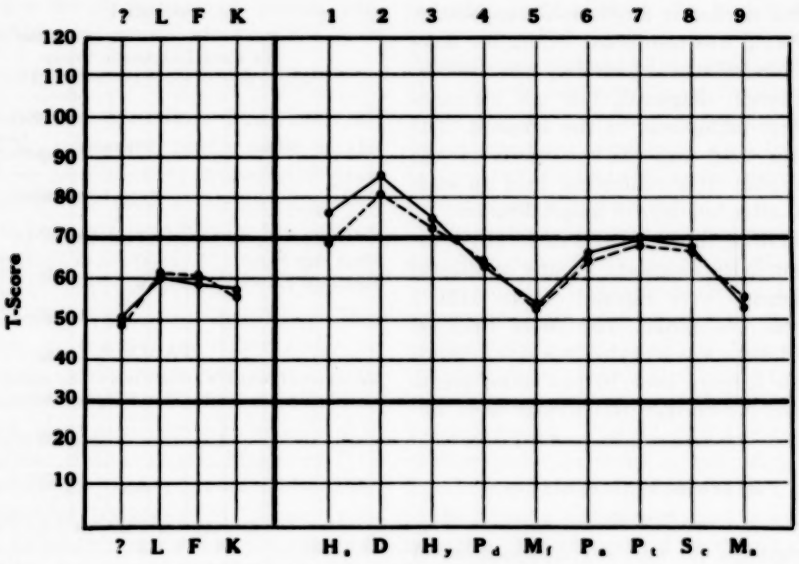

FIG. 3. MMPI profiles of male and female depressive cases.
—— Male depressives. Code: 2″13′7864-59 L-FK?.
- - - Female depressives. Code: 2″3′17864-95 LF-K/?.

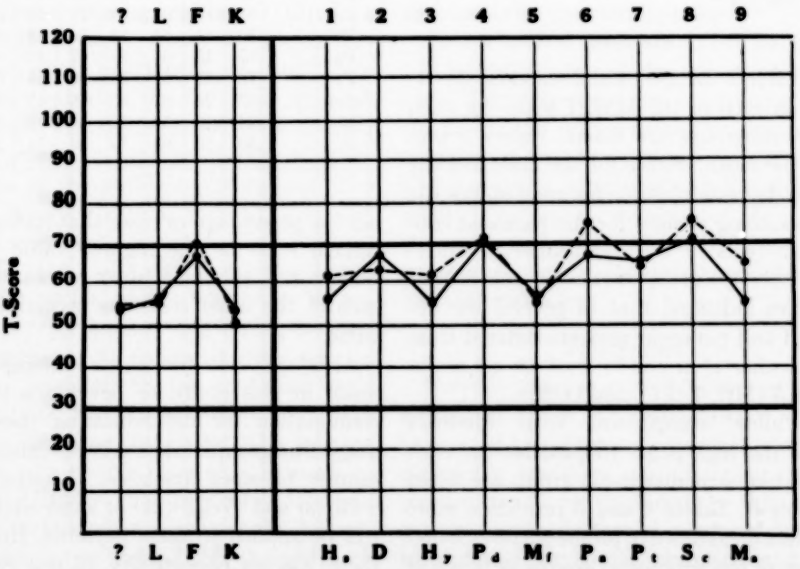

FIG. 4. MMPI profiles of male and female paranoid schizophrenic cases.
—— Male paranoids. Code: 48′267-51 39 F-L?K.
- - - Female paranoids. Code: 864′97 213-5 F′-L?K.

TABLE 3

Frequency of Each MMPI High Point In the Various Diagnostic Subgroups

MMPI High Point	Depression		Paranoid Schizophrenia	
	Males	Females	Males	Females
1 (Hs)	5	5	2	
2 (D)	10	17	2	6
3 (Hy)	2	2		4
4 (Pd)		3	4	8
5 (Mf)			1	1
6 (Pa)		2	1	9
7 (Pt)		1	1	1
8 (Sc)		2		3
9 (Ma)			2	3
0 (Si)		1	1	1
Totals	17	33	14	36

that are termed "floating profiles." He reported that the relationship between Hy (3) and Pd (4) was highly related to the direction of expression of emotional disturbances, particularly the manifestations of hostility. Where, that is, scale 4 exceeded 3, acting out was frequent and control was inadequate or sporadic. In his cases where 3 exceeded 4, however, the individual was characterized by a long series of neurotic complaints, fatigue, dysphoria, crying spells, and inadequacy. It would appear that scale 3 values reflect the level of control and inhibition; when the score on scale 4 exceeds the value of scale 3, direct expression of impulses appears to be highly probable.

TABLE 4

Per Cent of Each Pair of High Points in Codes from Depressives

Second Point	High Point										Second Point Totals
	1	2	3	4	5	6	7	8	9	0	
1		6									6
2	9		2	8		2	4	8			33
3	5	13									18
4		2	2			2					6
5			4								4
6				4							4
7		17						2			19
8		4				2					6
9			2								2
0		2									2
High Point Totals	14	44	10	12		6	4	10			100

TABLE 5

Per Cent of Each Pair of High Points in Codes from Paranoid Schizophrenics

Second Point	High Point										Second Point Totals
	1	2	3	4	5	6	7	8	9	0	
1			2.0			2.0					4.0
2	2.0		2.0	6.0		2.0	1.0	1.0			14.0
3				4.0		2.0					6.0
4		2.0				4.6		2.0	2.0	2.0	12.6
5										2.0	2.0
6	2.0			6.0	2.0			5.0	4.0		19.0
7	2.0			2.0				2.0	1.0		7.0
8		4.0		2.0	2.0	8.6	4.0		3.0		23.6
9				1.0	2.0	2.6	2.0				7.6
0		2.0		1.0			1.0				4.0
High Point Totals	6.0	8.0	4.0	22.0	6.0	21.8	8.0	10.0	10.0	4.0	99.8

TABLE 6

Relationship between Scales 3 (Hy) and 4 (Pd) in the MMPI Profiles of Each Diagnostic Group

MMPI Pattern	Group		Total
	Depression	Paranoid Schizophrenia	
$3 \geq 4$	39 (78%)	17 (34%)	56 (56%)
$4 > 3$	11 (22%)	33 (66%)	44 (44%)
Totals	50	50	100

$\chi^2 = 19.642$ ($p < 0.001$).

Since clinical manifestations of this configuration bear directly upon the two syndromes under consideration in this paper, it seemed appropriate to apply a similar analysis to these data. The results are shown in Table 6. The separation is highly significant.

DISCUSSION

The mean values of these hospitalized groups on the basic MMPI scales that are tabulated in Table 2 and plotted in Figures 3 and 4 show a high level of correspondence to the previous findings in the literature. This consistency is particularly clear in the profiles from the depressed groups. It should be noted that the configuration of the validity scales is unusual in the rise on L, either tied with or actually higher than the average value of F. The data from our paranoid groups are most similar to Wauck's paranoid schizophrenics (Figure 2) which they most clearly resemble in diagnostic composition as well. The paranoid group from Guthrie's study (Figure 2) is also quite similar to the present groups, particularly in level of elevation.

It is also clear that the two diagnostic groups give quite different score patterns on the MMPI, being discriminable on several scales in addition to the two criterion variables involved, scales 2 and 6. Inspection of the distributions of scores on each of the basic scales separately revealed, however, that there was large overlapping of these two groups and that no one scale gave a practically useful degree of separation for diagnostic purposes. It would appear however from the data in Tables 5 and 6 that a useful score combination could be advanced for such a differential diagnostic purpose.

From Table 3 it can be seen that 54 per cent of the depressed cases had their peak score on scale 2 as opposed to 16 per cent of the paranoid cases. This is not as large a percentage as that reported by Modlin in his depressed sample. Only 20 per cent of the paranoid cases had their peak score on scale 6, in contrast to the four per cent of the depressed cases. A slightly larger segment (24 per cent) of the paranoid cases had their highest score on scale 4 (Pd).

In Table 4 it can be seen that scale 2 was highest or second highest in the profiles on 77 per cent of the depressed group. Scale 2 is equally prominent in only 22 per cent of the paranoid sample. In the paranoid profile patterns, scale 6 is the first or second value in 41 per cent of the cases, while it is equally prominent in the depressed cases only in 10 per cent of the cases.

Hathaway and Meehl (12), in a tabulation of the nearly 1,000 cases in the *Atlas* by diagnostic status, found that the combination of depression and psychasthenia (code 27) was the single most frequent (26 per cent) score pattern in the 121 cases diagnosed psychotic depression. Other frequent patterns were: 72 (7.4%), 82 (7.4%), 23 (7.0%), and 28 (6.6%). In the present sample of depressed cases, the single most frequent high point pair was also 27, although only 17 per cent of the cases showed this pattern. Most prominent in the high point pairs here were the combinations of scale 2 with scale 1 (Hs) and with scale 4 (Pd).

Hathaway and Meehl also analyzed the patterns in 128 cases with a diagnosis of paranoid psychosis. They found a result quite similar to that shown in Table 5; that is, the high point pairs did not show the homogeneity of the depressed sample. The

most frequent pair they report was a code of 23 that appeared in 7.4 per cent of the cases. Other frequent patterns were 49 (5.5%), 48 (5.5%), and 68, 43, 46, 26, all with 4.3 per cent each. In Table 5 it can be seen that 68 is the most frequent pattern but it appears in only 8.6 per cent of the test records. The reverse pattern of 86 accounts for another 5 per cent. The combination of scale 2 and 4 is also frequent in this group, as is 46.

The relationship between scales 3 and 4, even when they are not the prominent scales in the profile, would appear to be another useful index in differentiating these reactions and in understanding the personality processes involved. If hostile impulse expression is reflected in the height of scale 4 and one form of control over this expression is measured by scale 3, then the findings in Table 6 lend support to the view that a paranoid reaction pattern is accompanied by a lack of effective controls over such unacceptable behavior. Similarly, evidence of considerable blocking of these strong motives is found in the patterns of scales 3 and 4 in the depressive cases. The prominence, at the same time, of scale 7 values in the depressives suggests a turning inward of these hostile feelings in these cases. It should be noted, however, there are some glaring exceptions to these trends in the MMPI records obtained on these hospitalized patients. It should be rewarding to scrutinize the ward behavior of these cases to discover whether there had occurred any shifts in their behavior by the time the MMPI was obtained or whether there are significant variations in impulse expression from one MMPI subgroup to another within these broad diagnostic groupings.

SUMMARY

Fifty cases each of depressive psychotic reaction and paranoid schizophrenic reaction were examined with the MMPI within ten days after admission to a state hospital. The test findings were compared to group profiles of similar groups reported in the literature and a pattern analysis of the MMPI profiles was carried out. Some suggestions for differential diagnosis and for understanding the dynamic processes underlying these two psychotic reactions were advanced.

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DENIAL OF ILLNESS IN RELATION TO INTELLECTUAL FUNCTION

DAVID G. FRYER, M.B, Ch. B.[1] AND MARGARET P. RICH, Ph.D.[1]

INTRODUCTION

It has several times been reported that some patients with pulmonary tuberculosis react to the diagnosis of their disease by disbelief or denial (15, 18, 19). Our interest in the subject of denial arose out of an empirical observation made during routine psychiatric assessment interviews with new patients at the Boston Tuberculosis Sanatarium. It was noted that those patients who deny their disease or its serious implications tended to have more difficulty in sentence repetition than other patients. This observation was substantiated in a pilot study on 53 patients. It was assumed that the ability to repeat sentences dictated by the interviewer was a rough measure of intellectual level and the hypothesis was made that the use of denial as a defense mechanism occurs most often in people with impaired or low level of intellectual function. This present study is designed to test this hypothesis.

PROCEDURE

The subjects were male patients admitted to the Sanatarium in routine way over a period of five weeks. During this time 28 patients were admitted of whom 26 were included in this series. The two excluded were a transferee from a penal institution where he had been treated for tubercle for six months and who was therefore not a "new" patient, and a Chinese patient who spoke no English. The subjects were interviewed by a physician and the interviews recorded. No attempt was made in these interviews to apply intelligence tests. The interviewer categorized each subject as either a "denier" or a "non-denier." The patients were all white males who ranged in age from 31 to 66 for the "non-deniers" with a mean age of 49.1, and from 19 to 78 for the "deniers" with a mean age of 53.9. There was no significant difference in age for the two groups as tested by the Mann-Whitney U Test, a non-parametric procedure devised to compare small groups.

Two tests, the Babcock Sentences (1) and the Shipley-Hartford Retreat Scale (13) were administered by a clinical psychologist who was unaware of the patients' classification regarding denial of illness.

The Babcock Sentence test consists of twenty-three sentences which are progressively more difficult. This test, which measures recent memory function as it is demonstrated by the success in repeating these increasingly difficult sentences, was given individually to each patient. The Shipley-Hartford Scale, which consists of two parts, a multiple choice vocabulary test and an abstraction test requiring the completion of a series of number, word, or letter sequences, was given to patients in groups of five to seven.

Each of the subjects was subsequently seen by a social worker who was unaware of the results of the other two investigators. The social worker attempted to estimate the incidence of chronic alcoholism in the sample.

RESULTS

Babcock Sentence Test: The mean test score of the "non-deniers" on the Babcock Sentences was 13.1; the mean for the "deniers" was 10.3. The difference between the

[1] Department of Psychiatry, Harvard University School of Medicine; Boston City Hospital, 818 Harrison Avenue, Boston, Massachusetts. The authors gratefully acknowledge the cooperation of Mr. J. Scott, Medical Social Worker, Boston Tuberculosis Sanitarium, in gathering data for this report.

TABLE 1
Data for All Subjects

Non-deniers							Deniers						
	Age	Babcock Sentences	Vocabulary Score	Abstraction Score	Mental Age	Conceptual Quotient		Age	Babcock Sentences	Vocabulary Score	Abstraction Score	Mental Age	Conceptual Quotient
1	36	14½	18	4	9.9	71	1	66	3	—	—	—	—
2	54	12½	16	8	10.2	86	2	60	9½	13	2	8.7	78
3	53	12	22	8	11.2	71	3	52	12	18	14	11.5	92
4	66	16½	24	6	11.2	63	4	19	16	18	10	10.9	84
5	46	11	22	10	11.5	75	5	78	9½	24	2	10.6	56
6	31	11	28	6	11.9	57	6	43	15	28	10	12.7	64
7	62	15½	25	10	12.1	68	7	52	11	21	6	10.7	69
8	41	12½	26	10	12.3	67	8	46	8	14	6	9.5	82
9	59	10	32	6	12.7	53	9	51	10½	17	6	10.0	78
10	32	11	26	20	14.3	83	10	72	8	14	2	8.9	75
11	50	14	35	12	14.5	62							
12	60	11	30	20	15.1	77	n	10	10	9	9	9	9
13	34	14½	32	20	15.5	73	$\bar{X}$	53.9	10.3	18.6	6.4	10.4	75.3
14	50	15½	31	24	16.1	81							
15	52	14	30	28	16.7	92							
16	59	14	39	26	17.9	84							
n	16	16	16	16	16	16							
$\bar{X}$	49.1	13.1	27.3	13.6	13.3	72.7							

two groups was evaluated by the non-parametric Mann-Whitney U test, which compares the rank orders for the two groups (9). The score rankings gave a U value of 37.5, yielding a p of $<.025$.

Shipley-Hartford Scale: This scale has been considered to be a reasonably valid measure of intelligence (8, 10), and has been used for this purpose. There are four measures derived from the Shipley-Hartford scale: the Vocabulary Age, which is derived from the vocabulary scale score; the Abstraction Age, derived from the abstraction scale score; the Mental Age, obtained from the *summation* of the vocabulary and abstraction scale scores; and the Conceptual Quotient, which is calculated from the *ratio* of the Abstraction Age to the Vocabulary Age. This quotient purports to measure organic impairment.

The two groups showed a significant difference in Mental Age. The mean M.A. for "deniers" was 10.4, while the mean M.A. for "non-deniers" was 13.3. The U value of 20 for the score rankings produced a p value of $<.001$.

The following statement is made in the manual of directions. "The validity of the total Mental Age is related to the size of the C.Q. If this latter is normal, the indicated Mental Age is probably valid. If impairment is revealed in the quotient, the Mental Age merely represents a level midway between the patient's best and least preserved capacities; perhaps it strikes a rough average of his present abilities" (13, p. 3). Conceptual Quotients below 100 are considered to be in the direction of impairment.

The Conceptual Quotients for the two groups did not differ significantly. The mean C.Q. for "non-deniers" was 72.7; the mean for the "deniers" was 75.3. Inspection reveals that only one subject in each group obtained a C.Q. of 90 or above, which is designated as "normal" by the authors of this test. All other subjects obtained C.Q.'s to which some degree of suspicion of pa-

thology is attached. A literal interpretation of the test would lead to the conclusion that some degree of deterioration may have occurred in most of the patients in *both* groups. However, the Manual of Directions cautions, "The available evidence indicates that the C.Q.'s obtained from subnormals are not valid. Feebleminded and borderline cases tend to earn low quotients. For this reason, *extreme caution* must be observed in interpreting quotients from individuals with vocabulary scores below 23 (14 years)" (13, p. 3).

Using this criterion for categorizing the subjects, there were four "non-deniers" whose vocabulary scores were less than 23, and 12 whose scores were above 23. In contrast, there were eight "deniers" whose vocabulary scores were below 23, and two whose scores were above 23. The mean vocabulary score for "non-deniers" was 27.3; the mean score for "deniers" was 18.6. The U value for the two groups was 19, giving a highly significant p value of $<.001$.

The abstraction scores for the two groups were also significantly different, although the difference was less marked than that for the vocabulary scores. The mean abstraction score for the "non-deniers" was 13.6, and the mean for the "deniers" was 6.4. The score rankings gave a U value of 32.5, which yields a p value of $<.01$.

These results show that the "non-deniers" were superior to a statistically significant degree in repeating the Babcock Sentences. They had a significantly higher Mental Age as determined by the Shipley-Hartford Scale, and scored significantly higher on both its sub-tests, the vocabulary scale and the abstraction scale. The superiority was much more marked for performance on the vocabulary scale. Both groups obtained Conceptual Quotients which were in the "suspicious" range, and the two groups did not differ significantly.

Report of Cases: Various phenomena were observed in those ten subjects regarded as "deniers." Two patients showed complete denial in the sense that they did not accept that they were ill in any respect. One of these rationalized his presence in hospital by saying he had been sent there for nothing by a friend who was stupid. The other man claimed his doctor had sent him for a rest and when pressed further added a confabulation about how the resident in charge of his case had told him he could go home tomorrow.

Two patients claimed not to know the reason for their being in hospital and made no attempt to get information from the examiner in spite of opportunity and encouragement to question him.

In another case there occurred a defense which may be described as isolation from responsibility, in which the patient detached himself from the unpleasant connotation of his illness by delegating them to a care of another person. When asked if he had inquired why his son had arranged for him to go to hospital this patient replied, "I didn't ask him, no. It was up to him."

In another patient we observed an attribution of the hospitalization to a less threatening illness. This man was initially admitted to another hospital. Here tuberculosis was diagnosed and the patient subsequently developed stiffness of his shoulders which was described as "functional" by a neurologist. When asked during our interview what concerned him most he replied that it was his "arthritis," and went on to say, "If this arthritis were cared for I'd be out in three days."

Four patients attributed the symptoms of their illness to a benign cause. For example, loss of weight, weakness and wasting were attributed to arduous working conditions by a man who said he was suffering from "only the T.B."

A noticeable feature of the denying patients was the paucity of expression of anxiety or depressive ideas. Such statements as, "Mentally I feel lower than a snake's belly" were made by the "non-deniers" but not by the "deniers."

The social worker estimated that twelve men in this study were "alcoholics." Four of these were among the "deniers" and eight among the "non-deniers." (An "alcoholic" was here defined as one who drinks more than a heavy social drinker and who cannot control his intake of alcohol.)

DISCUSSION

To the extent that it may be assumed that vocabulary functions remain relatively intact in the face of brain damage it would be necessary to consider the likelihood that most of the "deniers" were cases of borderline intelligence to whom the determination of C.Q. could not validly be applied; this would follow from the criterion of vocabulary level specified in the test manual.

Williams *et al.* (17) question the assumption that vocabulary can be assumed to be relatively unimpaired, and as a result of their findings conclude, "Traumatic brain injury results in a general deficit in intelligence test scores with little differential deficit .... These results, together with Yates' (1956) penetrating analysis, should end the myth that verbal tests such as Reading and Vocabulary are resistant to the effects of brain injury" (17, p. 304).

Parker (10) reports a study in which the Shipley-Hartford Scale was used to test a group of 30 patients with diagnosed brain damage and a control group of 30 hospitalized patients not suspected of having brain damage. He reports results similar to those found in this study: the brain-damaged patients had a mean C.Q. of 88.3, while the mean of 80.3 for the control group was slightly, albeit not significantly, lower (*i.e.*, more in the direction of suspected brain damage). Parker points out that in "studying only a brain injured group and a normal group, it has often been assumed that obtained differences were due to the brain injury ... There is the possibility that the obtained differences may be due to any organic condition and, therefore, found not only in the case of S's with brain damage" (10, p. 425).

It is clear that Parker's control group, as well as the groups here reported, deviated strikingly from the pattern of the standardization sample. Both the "denier" and "non-denier" groups in the present study showed general impairment of function; it was not possible to determine whether this was a function of physical illness, of possible brain damage, or whether in some instances it was a matter of limited native intellectual endowment.

All the "deniers" and, in fact, most of our "non-deniers" too, were people who performed rather poorly on the intelligence tests, the "deniers" performing more poorly than the "non-deniers." But the tests did not distinguish between low inherent intelligence and intellectual impairment secondary to say, chronic alcoholism or senile and arteriosclerotic change.

Denial of illness has been well documented in cases of organic brain disease. Weinstein and Kahn (16), in their study of such patients, state that those showing explicit verbal denial have outstanding premorbid personality characteristics but the organic brain damage is necessary to create a state of brain function in which patterns of denial may operate.

On the other hand, as is described by Fenichel (2), denials which are not at all supported by organic impairment may occur in psychoneurotic patients. Where do the cases of denial in our study fit in between these two extremes?

The subjects in this study consisted of consecutive routine tuberculosis first-admission cases. These patients were mainly from the lower socio-economic class of Boston. It is common experience (5, 6, 12, 15) that tuberculosis hospital admissions include a large number of alcoholics and "skid-roaders," and it has been stated that denial is a characteristic of the so-called alcoholic personality (4, 7). In our study there was no

difference in the proportion of alcoholics in the "denier" and "non-denier" groups.

It is no doubt significant that our patients suffered less obvious disablement than those of Weinstein and Kahn (who denied hemiplegia, blindness and pain) so that denial in our subjects required a smaller sacrifice of the awareness of reality. Fenichel (2) points out that "only in the case of severe disturbance of the function of reality testing (psychoses) do important denials remain victorious in adults." Denial is characteristically a defense seen in children. Anna Freud (3) states that this mechanism belongs to a normal phase in the development of the infantile ego and Sandifer (11) makes a similar observation in relation to anosognosia of blindness in children.

It appears that our patients with less intellectual ability react like the child unable to take flight from a "painful" situation. Where this low intellectual ability is an index of poor endowment it may be supposed that the availability of the defense of denial has persisted and in certain kinds of brain damaged cases there appears a regression to an earlier, more primitive level of ego function.

CONCLUSIONS

Twenty-six unselected early tuberculosis patients were examined to determine whether or not the occurrence of denial of their illness was related to level of intellectual function.

The patients were tested by the Shipley-Hartford Scale and the Babcock Sentence tests. Ten patients denied their illness and it was found that denial was significantly related to lack of ability to perform the tests.

It was discussed whether the low level of intellectual function was due to low native intelligence, organic impairment or to sickness and hospitalization. We cannot state which of these factors is the most important and perhaps they were operative to varying degrees in different subjects.

The results demonstrate that low level of intellectual function predisposes to and is positively related to the evocation of the defense of denial.

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DRUG-ACTION PROFILES BASED ON AN ABBREVIATED PSYCHIATRIC RATING SCALE

DONALD R. GORHAM, PH.D.[1] AND JOHN E. OVERALL, PH.D.[1]

The large number of drug evaluation studies since the advent of tranquilizers is evidence of a crucial need for efficient methods of assessing changes in psychiatric condition. Frequent failures to identify differential patterns of response to drugs which are quite different chemically emphasizes the need for improved measuring techniques. Increased efficiency can be expected of instruments designed specifically to measure *change* in a *selected type of patient* during treatment with a *specified class of drugs*.

The present writers have been in a particularly favorable position to work on the development of such instruments, having had available for analysis psychiatric ratings on a large number of patients from chemotherapy investigations of the Veterans Administration Cooperative Studies in Psychiatry.[2] The study from which the data reported here were taken was a double-blind evaluation of the relative therapeutic effectiveness of six tranquilizing agents. Approximately 475 newly admitted male schizophrenic patients were randomly divided among the six treatment groups. The Multidimensional Scale for Rating Psychiatric Patients (MSRPP), developed by Lorr (2), was employed to evaluate the psychiatric condition of each patient before, during and after the 12-week treatment period. A detailed report of the study has been presented by Casey, Lasky, Klett and Hollister (1).

METHOD AND PROCEDURE

Pre- and post-treatment scores on each of the 40 items of the psychiatric interview portion of the MSRPP were were compared for each patient, and a score of 1 or 0 was assigned, depending upon whether the ratings indicated improvement or failure to improve, respectively.

Sums of squares and cross-products of these 40 scores were computed for each drug group separately, corrected to the drug-group means, and then summed over groups. That is, the sums of squares and cross-products were computed from deviations of the individual scores about the means for the treatment groups to which the individual belonged. A "within-groups" matrix of intercorrelation was computed by a standard product-moment formula from the corrected sums of squares and cross-products.[3]

The matrix of intercorrelations between change scores on the 40 items was subjected to cluster analysis. Nine relatively independent clusters were identified. The two items with highest average intercorrelation with other items in each cluster were selected for inclusion in a tentative abbreviated rating scale composed of 18 items.

After reversal of appropriate items, the correlation between "total score" on the 18-item scale and "total score" on the full 40 items of the psychiatric interview portion of

[1] Veterans Administration Central Neuropsychiatric Research Laboratory, Perry Point, Maryland.

[2] The Cooperative Studies in Psychiatry have included a series of large-scale double-blind drug-evaluation studies involving dozens of Veterans Administration Hospitals throughout the country. Primary credit belongs to the scores of psychiatrists and psychologists who obtained the data analyzed here. The results of previous studies are summarized in the Transactions of Research Conferences on Chemotherapy in Psychiatry (1957, 1958, 1959, 1960), Office of the Chief Medical Director, Veterans Administration, Washington 25, D. C.

[3] A standard product-moment program was available for the electronic computer used in the calculation of results. Although this method is only approximately correct for the dichotomized scores, it is considered adequate for the purpose of cluster analysis.

the MSRPP was computed. The validity of the short rating scale as a substitute for the longer scale in evaluating drug effects was determined by computing the correlation between change scores on the two measures based on the original sample of 475 acute schizophrenic patients. A cross validation of the findings was secured by comparing the short scale with the longer one in a six-month study by Shawver, Gorham, Leskin, Good and Kabnick (4) in which Thorazine and Serpasil were used with 120 chronic schizophrenic patients.

Since the correlations were high enough to demonstrate that the short form was substantially equivalent to the longer one (of Table 3), the nine sub-scales were applied to the six drug groups to determine whether these sub-scales which were developed from change scores could separate drug groups as well as the Lorr factor scores, or better.

RESULTS

From the two central items in each cluster, an 18-item abbreviated scale was constructed and tentative names assigned to the nine symptom areas represented. Seven of the pairs of items representing discrete symptom areas are found in seven different scales in the original Lorr factor keys. The remaining two pairs each include items from two different Lorr factors. Table 1 shows the location of the 18 items of the drug change scale in the original Lorr factors. Although the two items in a single change cluster are found in two different Lorr factors in only two cases, in several other cases the change-sensitive items are imbedded in a larger group of items in such a way that the interpretation of the factor is different from what it would be for the change-sensitive items alone. Perhaps these differences are responsible for the greater effectiveness of the change-sensitive items in discriminating between drug effects (see below).

Table 2 is a list of the 18 items selected from the results of the cluster analysis of intercorrelations between change scores on the psychiatric interview portion of the MSRPP.

The next step in the investigation involved going back to the original data and computing change scores for each of the nine symptom areas separately and in combination for each patient. This was done by summing together the pre-treatment ratings

TABLE 1

Relationships of Change Factors to MSRPP Status Factor

Change Factor	Item Numbers	Location of Items in MSRPP Factor* Configuration
1. Unwarranted suspicion	33, 37	C: 10, 33, 37
2. Hallucinatory behavior	22, 27	F: 15, 16, 22, 27, 38
3. Disorientation	15, 32	B: 32, 48 F: 15, 16, 22, 27, 38
4. Tension-anxiety	5, 17	E: 5, 17, 21, 23, 34, 36, 40
5. Inaccurate self-concept	25, 30	J: 6, 25, 30
6. Emotional responsiveness	31, 36	A: 8, 20, 23, 31, 34, 36
7. Conceptual disorganization	1, 11	K: 1, 3, 4, 11, 28, 29, 38
8. Mannerisms, posturing	2, 9	G: 2, 9, 15, 18, 20, 28, 29
9. Guilt and dread	6, 40	J: 6, 25, 30 E: 5, 17, 21, 23, 34, 36, 40

* The names assigned by Lorr to his 12 factors based on the full 62 item scale are:

A. Retarded depression vs. manic excitement
B. Compliance vs. resistiveness
C. Paranoid projection
D. Activity level
E. Melancholy agitation
F. Perceptual distortion
G. Motor disturbances
H. Submissiveness vs. belligerence
I. Withdrawal
J. Self-depreciation vs. grandiose expansiveness
K. Conceptual disorganization
L. Hysteric conversion

TABLE 2

Abbreviated MSRPP For Evaluating Drug Change

1. Unwarranted Suspicion

33. Does he tend to suspect or to believe on slight evidence or without good reason that some people are against him (persecuting, conspiring, cheating, depriving, punishing) in various ways?

37. Does he tend to suspect or to believe on slight evidence or without good reason that some people talk about, refer to, or watch him?

2. Hallucinatory Behavior

22. Does he report or admit to hearing hallucinatory sounds or voices? To what extent does he believe in their independent existence?

27. Does he report or admit to seeing hallucinatory images of objects or persons? To what extent does he believe in their independent existence?

3. Guilt and Dread

6. Does he tend to feel or to believe on slight evidence or without good reason that he is unworthy, sinful, evil, or guilty?

40. Does he admit or report a feeling of fear about vague unknown events which may take place in the future? Or does he anticipate without good reason, tragic, inescapable misfortunes or disasters?

4. Disorientation

15. Does he give evidence of (A) knowing who he is, (B) recognizing several persons about the hospital, (C) knowing the names of several persons around the hospital?

32. How well oriented is he as to where he is? Does he know (A) that he is in a hospital; (B) the state, or the city nearest the hospital; (C) the name of the hospital?

5. Tension-Anxiety

5. Dose he appear to be characteristically placid and relaxed, or tense and "nervous"? Consider such signs as fidgeting, tremors, muscle twitching, body rigidity, and sweating.

17. Is he, or does he easily become, concerned, uneasy, or apprehensive to a degree not called for by external circumstances?

6. Inaccurate Self-Concept

25. Consider how favorably he appears to regard himself. Does he tend to be self-depreciatory (feel inferior, inadequate) or conceited (feel self-assured, important, superior)?

30. Does he have an exaggeratedly high opinion of himself or an unjustified belief or conviction of having unusual ability, knowledge, power, wealth, or status?

7. Emotional Responsiveness

31. Compared to others, is he controlled, restrained, inhibited and unable to show feeling, or is he more inclined to be unrestrained, uncontrolled, or free in showing feeling?

36. How emotionally responsive does he appear to be? Does he tend to show an under-reaction or an over-reaction to situations?

8. Conceptual Disorganization

1. How direct and relevant are his responses to questions or to the topic discussed?

11. Are the elements of his speech logically consistent and connected by some idea or relationship, or do they tend to be inconsistent and disconnected? Rate what is most representative during the interview.

9. Mannerisms, Posturing

2. Does he assume or maintain peculiar, unnatural, or bizarre postures?

9. Does he exhibit any repeated peculiar inappropriate gestures, grimaces, or manneristic movements? (Do not confuse with compulsive acts which the patient recognizes as irrational or absurd.)

TABLE 3

Intercorrelation of Change Factors and Total Scales

	1	2	3	4	5	6	7	8	9	10	11	12
1	1.00	0.39	0.01	0.32	0.14	−0.08	0.31	0.25	0.37	0.70	0.66	0.60
2	0.39	1.00	0.18	0.22	0.11	−0.02	0.26	0.28	0.28	0.68	0.59	0.55
3	0.01	0.18	1.00	0.06	−0.02	0.02	0.26	0.26	0.10	0.35	0.31	0.39
4	0.32	0.22	0.06	1.00	−0.14	−0.14	0.24	0.27	0.40	0.53	0.49	0.45
5	0.14	0.11	−0.02	−0.14	1.00	−0.03	0.18	−0.01	−0.14	0.26	0.23	0.11
6	−0.08	−0.02	0.02	−0.14	−0.03	1.00	0.03	−0.04	−0.02	0.17	−0.05	0.01
7	0.31	0.26	0.26	0.24	0.18	0.03	1.00	0.43	0.22	0.65	0.63	0.62
8	0.25	0.28	0.26	0.27	−0.01	−0.04	0.43	1.00	0.26	0.63	0.54	0.53
9	0.37	0.28	0.10	0.40	−0.14	−0.02	0.22	0.26	1.00	0.58	0.50	0.51
10	0.70	0.68	0.35	0.53	0.26	0.17	0.65	0.63	0.58	1.00	0.93	0.89
11	0.66	0.59	0.31	0.49	0.23	−0.05	0.63	0.54	0.50	0.93	1.00	0.84
12	0.60	0.55	0.39	0.45	0.11	0.01	0.62	0.53	0.51	0.89	0.84	1.00

1. Unwarranted suspicion
2. Hallucinatory behavior
3. Disorientation
4. Tension-anxiety
5. Inaccurate self-concept
6. Emotional responsiveness
7. Conceptual disorganization
8. Mannerisms, posturing
9. Guilt and dread
10. Total score on abbreviated scale (18 items)
11. Morbidity score: psychiatric interview portion (40 items)
12. Morbidity score on total MSRPP (62 items)

on the two items in a symptom area and then subtracting the sum of the post-treatment ratings on the two items.[4] Table 3 presents the intercorrelations between change scores in the nine symptom areas and the correlations of these change scores with change scores for the combined 18 items of the abbreviated scale and for the full 40 items of the psychiatric interview portion of the MSRPP. The correlation between the "total score" on the 18-item abbreviated scale and the "total score" on the 40-items from the MSRPP was found to be .93 for the sample of 474 acute schizophrenic patients. A correlation of .93 between the short and long forms was found in the cross validation sample of 120 chronic schizophrenics.

Since the new sub-scales were developed from analysis of change scores, the next step in the investigation involved evaluation of differences in the degree of change resulting from treatment with the different drugs used in Project 3 (1). The finding that the drug-change scales are sensitive in differentiating between patterns of specific drug effects can be viewed as being especially significant since the scores derived from the original MSRPP factor scales were relatively ineffective for this purpose. In particular, it would be gratifying to identify differing patterns of action for drugs within the phenothiazine group. Table 4 shows all differences between drug groups which reached statistical significance ($p < .05$).[5, 6]

Although the nine drug-change scales are not completely independent, the correlations between them are recognized to be reason-

[4] Bipolar scales for items in change clusters five and six presented a problem. Since most schizophrenics tend to be somewhat over-restrained or flat, the scoring of items in the "emotional responsiveness" area was reversed so that a higher difference score could be interpreted as improvement in agreement with other scoring.

[5] Because of the multiplicity of treatment comparisons, the true probability of a Type I error exceeds 5 per cent by a small amount for the comparisons in each symptom area. Nevertheless the arbitrary "5 per cent level" remains a reasonably adequate decision criterion.

[6] Statements regarding the relative effectiveness of the drugs must be interpreted within the framework of the drug dosage schedule which was set up by a committee of psychiatrists working with representatives of the drug companies. A fixed dose schedule was adhered to for the first four weeks of the study. During the last eight weeks, the physicians were instructed to adjust dosage, within limits, in an attempt to attain optimal therapeutic effectiveness for each patient.

TABLE 4

*Significant Differences Between Drug Effects in Nine Symptom Areas**

Unwarranted suspicion	Hallucinatory behavior	Disorientation	Tension Anxiety	Inaccurate self-concept	Emotional responsiveness	Conceptual disorganization	Mannerisms Posturing	Guilt and Dread	Total pathology
VESPRIN Phenobarb Pacatal	VESPRIN Pacatal Phenobarb	VESPRIN Phenobarb	VESPRIN Compazine Thorazine Phenobarb Pacatal	VESPRIN Phenobarb	VESPRIN	VESPRIN Pacatal Phenobarb	VESPRIN Pacatal Phenobarb	VESPRIN Compazine Pacatal Phenobarb	VESPRIN Thorazine Compazine Pacatal Phenobarb
TRILAFON Pacatal	TRILAFON Pacatal Phenobarb	TRILAFON Pacatal Phenobarb	TRILAFON Phenobarb Pacatal	TRILAFON Phenobarb	TRILAFON Compazine	TRILAFON Pacatal Phenobarb	TRILAFON Pacatal Phenobarb	TRILAFON Thorazine Compazine Pacatal Phenobarb	TRILAFON Thorazine Compazine Pacatal Phenobarb
THORAZINE Pacatal	THORAZINE	THORAZINE	Vesprin THORAZINE	THORAZINE Phenobarb	THORAZINE	THORAZINE Pacatal Phenobarb	Vesprin THORAZINE Phenobarb	Trilafon THORAZINE Phenobarb	Vesprin Trilafon THORAZINE Pacatal Phenobarb
COMPAZINE Pacatal	COMPAZINE	COMPAZINE	Vesprin COMPAZINE	COMPAZINE Phenobarb	Trilafon Pacatal COMPAZINE	COMPAZINE Pacatal Phenobarb	Vesprin COMPAZINE Phenobarb	Trilafon Vesprin COMPAZINE	Vesprin Trilafon COMPAZINE Pacatal Phenobarb
Vesprin Compazine Trilafon Thorazine PACATAL	Vesprin Trilafon PACATAL	Trilafon PACATAL	Vesprin Trilafon Compazine Thorazine PACATAL	PACATAL	PACATAL Compazine	Trilafon Thorazine Vesprin Compazine PACATAL	Vesprin Trilafon PACATAL	Trilafon Vesprin PACATAL	Vesprin Trilafon Thorazine Compazine PACATAL
Vesprin PHENOBARB	Vesprin Trilafon PHENOBARB	Trilafon Vesprin PHENOBARB	Vesprin Trilafon PHENOBARB	Vesprin Compazine Trilafon Thorazine PHENOBARB	PHENOBARB	Trilafon Thorazine Vesprin Compazine PHENOBARB	Vesprin Trilafon Thorazine Compazine PHENOBARB	Trilafon Vesprin Thorazine PHENOBARB	Vesprin Trilafon Thorazine Compazine PHENOBARB

* In each row one drug is shown (CAPITAL LETTERS) together with those other drugs whose actions differ from it: those above it are significantly better (0.05 level) and those below it are significantly poorer.

ably small. In this situation, it is informative to examine the profiles representative of the several drugs. In Figure 1 the mean change scores for the six drug groups are plotted as deviations (standard error units) about the "no change base line" for each of the nine sub-scales. The greater the improvement, the farther above the base line is the mean located (with the exception of the area concerned with "inaccurate self-concept," which should be conceived to run from "grandiose expansiveness" upward toward reduction in expansiveness and which could approach "self-depreciation" on one of the scale items).

From the "total pathology" scale, appearing at the extreme right of Figure 1, it is apparent that the overall effectiveness of Vesprin and Trilafon was significantly greater than that of the other drugs included in the study. In Table 4 it can be seen that no drug was significantly superior to either Vesprin or Trilafon in any symptom area. Although no statistically significant difference between Vesprin and Trilafon was found, differences in the observed profiles can lead to important hypotheses for further research. Further research may demonstrate that Trilafon is indicated for one type of patient and that Vesprin is indicated for treatment of another.

Although the overall effectiveness of Thorazine and Compazine was not found to be as great as the effectiveness of Vesprin and Trilafon, the overall effectiveness of these two drugs was found to be significantly greater than that of Pacatal and phenobarbital. Examination of the profiles of the former two drugs across the nine symptom areas reveals striking similarity in psychiatric effects. Only in the area of "emotional responsiveness" was any difference suggested, and even in this area, where Compazine produced a significant decrease in emotional responsiveness, no statistically significant difference between Compazine and Thorazine was found.

Pacatal and phenobarbital produced the least overall improvement in psychiatric condition. In the key schizophrenic symptom areas of "disorientation," "inaccurate self-concept," "conceptual distortion" and "mannerisms and posturing," phenobarbital was significantly worse than the four more effective phenothiazines. The effectiveness of Pacatal was significantly below that of the other phenothiazines in areas of "unwarranted suspicion" and "tension-anxiety."

The relative effectivenesses of the several drugs was consistent from one symptom area to the next. In general, the effective drugs (Vesprin and Trilafon) were effective in all areas, and the ineffective drugs (Pacatal and phenobarbital) were ineffective in all areas. One exception to the generality of drug effectiveness appears to be worthy of notice. There is a suggestion that Pacatal was comparatively less ineffective in improving emotional responsiveness and that Compazine had decreased effectiveness, *relative to the other drugs*, in this area. Actually, Compazine was found to produce a significant decrease in emotional responsiveness during the treatment period. One might suspect that the more potent tranquilizing agents would also be the ones found to have the greater stupefying effect; however, the rank ordering of drug group means in the emotional responsiveness area does not support this hypothesis.

DISCUSSION

The present investigation is a follow-up of earlier work (3) in which two higher order change factors were derived from relationships between change-scores on the MSRPP factor scales. One factor, which was identified with improvement in mental processes, contained MSRPP factors based upon items from the psychiatric interview. Since this factor was found effective in separating drug groups which were not previously found to differ significantly on the original MSRPP factor scales, a more detailed analysis was undertaken using only the psychiatric interview data and directing attention to the

* In each row one drug is shown (CAPITAL LETTERS) together with those other drugs whose actions differ from it: those above it are significantly better (0.05 level) and those below it are significantly poorer.

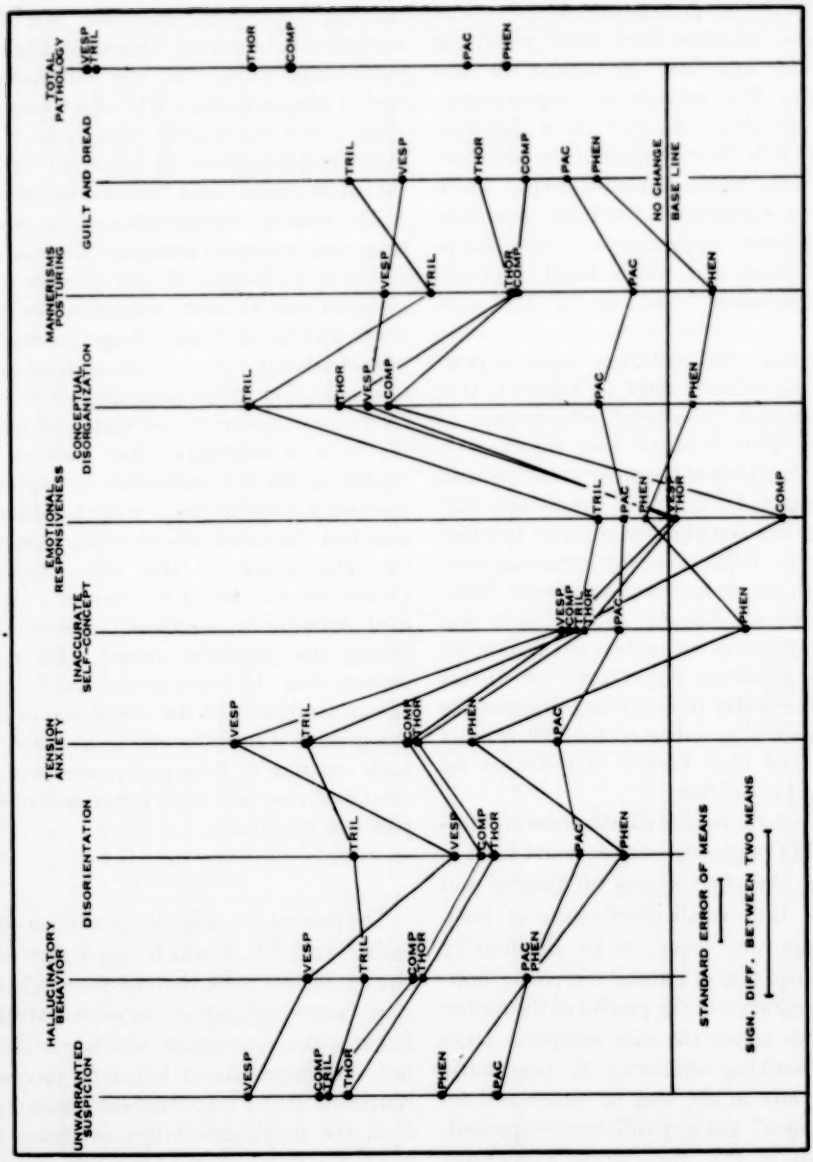

Fig. 1. Drug Action Profiles

problem of finding a limited number of items which are especially sensitive to change. The results have provided additional information concerning significant differences between drug effects, and a brief scale for rating drug change in psychiatric patients has been suggested.

Several approaches to the problem of identifying a small number of sensitive scale items were considered. A regression or discriminant function model might have been employed to select those items which yielded maximum separation between drug groups. This approach was not adopted because of the possibility of "capitalizing on chance relationships" when a larger number of items are considered initially. The present approach was adopted because it permits selection of a limited number of relatively independent symptom areas that are sensitive to change, before differences between treatment groups are even considered. This approach is basically quite different from one involving selection of items because of their observed effectiveness in discriminating between groups. The approach is especially useful where the number of variables under consideration is too large for treatment in a single regression analysis. Perhaps the most important outcome of the present analysis is a strong recommendation for the general approach to the problem involving 1) use of the "within-groups" correlation matrix which is independent of differences between treatment groups, 2) identification of relatively independent sources of variance, and 3) examination of the profiles of drug effects across the realtively independent symptom areas.

Another outcome of the present investigation is a brief drug-change key for the MSRPP or an abbreviated rating scale which can be used independently in research similar to that from which the data for the present investigation were taken. The efficiency of the brief scale depends upon the inclusion of items which are especially sensitive to drug change, the limited number of items used, and the possibility of the investigator's keeping in mind the nine discrete symptom areas and centering the interview about these areas. For research involving markedly different kinds of drugs or a different psychiatric population, it is recommended that extensive data be collected and reduced to change factors derived from the "within-groups" correlation matrix before treatment differences are examined.

SUMMARY

A short psychiatric rating scale, sensitive to changes found in acute schizophrenic patients treated with phenothiazines was evolved by cluster analysis from a correlation matrix based on a sample of 475 patients in a chemotherapy study.

When applied to the original sample, the new 18-item short scale correlated .93 with the longer 40 item MSRPP scale. This high validity held up when applied to a cross validation sample of 120 chronic schizophrenics.

The total score on the new short scale was more effective in separating drug groups than the "total morbidity" score of the full MSRPP.

The nine sub-factors of the short scale can be used to plot drug-action profiles which compare the specific action of drugs.

It is suggested that a nine-or-ten-item scale is feasible which would be especially useful in the rapid screening of new drugs.

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EFFECT OF LYSERGIC ACID DIETHYLAMIDE (LSD-25) ON IMPULSE CONTROL[1]

H. ARONSON, PH.D.[2] AND G. D. KLEE, M.D.[2]

INTRODUCTION

Intellectual Impairment: Lysergic acid diethylamide (hereafter LSD-25) was originally described as having an unusual and immediately striking effect upon perception. As the study of LSD has progressed from the descriptive, clinical level to a more rigorous, experimental stage, however, a number of investigators have also reported a detrimental effect of LSD upon intellectual ability. As examples, in 1955 Jarvik *et al.* found a reduction in the speed of performance on a test of simple arithmetic (3); and, Levine *et al.* found that LSD-25 significantly influenced all but two of the Wechsler-Bellevue Intelligence Scale subtests (5). In 1958 Silverstein and Klee reported a reduction in memory and an impairment of abstract reasoning ascribable to the drug (13). More recently, Silverstein and Klee found that a deficit in digit span (immediate memory of numbers) performance under LSD-25 could be demonstrated (12).

It is becoming increasingly evident that a change in intellectual functioning is brought about by the drug such that capacity is lowered but by no means completely destroyed. Another factor, the ability to utilize that rather considerable degree of intellect which remains, has been left largely uninvestigated.

Motivational Impairment: Recent studies of the changes brought about through psychosurgery have shown that one function especially sensitive to frontal lobe damage is the ability to foresee the consequences of an action and to modify behavior accordingly (10, 14). Basically, this is the ability to regulate behavior in accordance with the demands of reality, as opposed to directly attempting to satisfy drives as they develop. The Porteus Maze Test has had a long history of application in the measurement of this function. It is described by Porteus as a test of "social adaptability" or the ability both to inhibit impulsive solutions and to execute critical planning toward the achievement of goals.

Validation of this claim had been demonstrated by means of a number of studies based largely upon two fundamental designs. The first of these was to show that the test differentiated between individuals of equivalent general intelligence and comparable social environments, giving lower scores to those who had histories of sociopathic behavior. The second validation technique was to show that the test could significantly differentiate those individuals who were able to make adequate social adjustments out of groups considered subnormal by standard intelligence testing (8). The consistent findings that the Porteus Maze is one of the rare tests which is affected by frontal trauma (4, 6) not only helps in substantiating the test's validity, but also suggests that it may measure an extremely sensitive function.

The authors have observed clinically that there are some features of the LSD reaction which resemble the effects of prefrontal lobotomy. We refer specifically to motivation and behavioral control. Frequently observed in both states are reactions which show a basic indifference to ordinary environmental events. Coupled with this is a tendency to poorly planned, impulsive action. In both states it is often necessary to increase external control or supervision as a substitute

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[2] The Psychiatric Institute, University of Maryland School of Medicine, Baltimore, Maryland.

for control from within which the patient is less able or willing to achieve. We do not suggest that the many and highly complex effects of LSD can be explained simply on the basis of alteration of frontal lobe function. It is apparent, however, that some motivational change is brought about through LSD which parallels the effects of frontal surgery.

Since the Porteus Maze is purported to measure such a change and since it apparently does so in the case of frontal surgery, the test was chosen to evaluate the state of the function under LSD. It was our hypothesis that LSD, through its interference with critical judgment, would impair performance on the Porteus Maze Test.

PROCEDURE

The subjects were 68 male volunteers considered to be within normal limits on physical examination, psychiatric interview, and group psychological testing. They were randomly divided into a control group of 34, tested under normal conditions, and two experimental groups of six and 28, tested under LSD-25. In the drug group, six subjects were tested two to three and one-half hours after ingestion of 100 mcg. LSD-25; 28 were tested one and one-quarter to two and one-quarter hours after ingestion of 75 mcg. LSD-25. Placebos were not given for the control condition since facilities were not available to duplicate the care given the subjects in the drug group. Thus the two conditions differed in both the environmental atmosphere prior to the initiation of testing, and in the awareness of the subjects of the fact that they had (or had not) been asked to drink an unknown liquid.

The subjects were tested on the Vineland Revision of the Porteus Maze Test by individual administration. The Adult presentation of the Porteus consists of 10 paper-and-pencil mazes graded for difficulty from very simple to complex. The subject is asked to trace a path through each maze; unlimited time is given. If a mistake is made, however, the maze is immediately removed from view and the subject is asked to begin again, using a fresh copy of the maze. The subject is given a limit of either two or four trials, depending upon the complexity of the maze, to complete each pathway. The final score, stated in terms of an age scale, is determined by the number of incorrect trials taken on each of the mazes. These age scores, with an upper limit of 17 years, were the data for this study.

RESULTS

Before examining the results of the Porteus, since each subject was not compared with himself, it should be shown that the control group was sufficiently similar to the LSD group to have approximated the latter's performances under normal conditions. As stated above, the groups were randomly selected. The age and education of the two groups have been compared as an indication of the adequacy of the match.

As shown in Table 1, the experimental group is slightly older and more educated than the control group. Upon assumption of homogeneity of variance, t tests were performed to check the significance of the differences (2). The t's were: for age, .3622, for education, 1.3939. Neither is significant and the difference in education would, in any case, favor the experimental group.

A comparison of the Porteus scores, as seen in Table 2, shows the control group's scores to be superior to those of the 75 mcg. LSD-25 group which, in turn, are superior to those of the 100 mcg. LSD-25 group.

TABLE 1

Age and Education of the Control and Experimental Subjects

	N	Age			Education		
		Mean*	Median	Range	Mean*	Median	Range
Control	34	22.6	21	18–40	11.4	12	8–14
Experimental	34	23.0	22	18–39	11.9	12	9–16

* Difference not significant.

TABLE 2
Porteus Age Scores Under Non-Drug and Drug Conditions

	N	Porteus Age Scores		
		Mean	Median*	Range
Control	34	16.27	16.5	13–17
75 mcg.	28	15.21	16.0	10–17
100 mcg.	6	14.25	14.25	13–16

* All differences significant, $p < 0.05$.

These differences were analyzed by means of the Mann-Whitney Test (11) upon rejection of the assumption of normality and homogeneity of variance. Each of the differences was upheld by a significant Mann-Whitney *z*: control *vs.* 75 mcg. group, $p < .005$; control *vs.* 100 mcg., $p < .0005$; and 75 mcg. *vs.* 100 mcg., $p < .05$.

An additional analysis was performed to determine whether the Porteus was actually measuring ability to restrain behavior rather than merely reflecting a lower level of intellectual functioning. The variability of performance, *i.e.*, the number of mazes between the first failure and the last success, was tabulated for each subject. For example, if the first error were made on Maze V and the last adequate performance on Maze IX, the subject's range of response would be four mazes. A low variability score would suggest that the subject performed as well as he was intellectually able. Errors could be assumed to be due to an inability to perform as the subject reached the more difficult tasks. A high variability score, on the other hand, would indicate that the subject had available a greater intellectual capacity than he was utilizing, since he demonstrated this ability by succeeding with more difficult items. Thus at least a portion of credit lost by a subject whose variability was high could be ascribable to something other than intellectual deficit.

Table 3 shows the results of an analysis of the variability of performance of our experimental groups. As expected from the hypothesis, the drug group scores are higher than those of the control group. The probability of such a difference occurring by chance alone is, as determined by the Mann-Whitney Test, well below the one per cent level ($p < .005$).

DISCUSSION

The results are consistent with the clinical observation of a detrimental influence of LSD-25 upon the ability to exercise critical control over behavior. The highly significant difference between normal functioning and that obtained with only 75 mcg. would indicate that "anticipatory planning" is a function which is extremely sensitive to the action of LSD-25; and it is likely that considerably milder doses than those studied may be sufficient to produce a change.

Only one other drug, chlorpromazine, has been reported as showing an effect on maze performance. Like the results found here, the effect appears to be detrimental. Porteus and Barclay stated that "repeated administrations of the Maze Test to chronic psychotic patients receiving chlorpromazine, in comparison with a control group, reveal a continued deficit of about two years" (9, p. 299). This loss is comparable to that of our 100 mcg. subjects.

Thus, we are presented with a paradox of similar reactions under both a disorganizing and a therapeutic treatment. A possible explanation for this might be that chlorpromazine, in lessening the motivational strength of psychotic goals, also reduces the reality based striving necessary to inhibit some impulsive behavior. Porteus (6,7) noted an apparent indifference to frustra-

TABLE 3
Variability of Performance Under Non-Drug and Drug Conditions

	N	Variability Scores		
		Mean	Median*	Range
Non-Drug	34	1.71	1.5	0–4
Drug	34	2.97	2.0	0–8

* Difference significant, $p < 0.005$.

tion after repeated chlorpromazine medication which he considered in many ways similar to that seen in subjects with frontal surgery. Again, there is little additional evidence that chlorpromazine acts specifically upon the frontal or fronto-thalamic structure of the CNS.

The results of this study add no definitive information on the problem of localization of the function measured. Although these results resemble those found with lobotomy, they may also resemble the influence of a comparable loss or series of losses elsewhere in the brain. Very little is known about Porteus performance after damage in other areas. As mentioned earlier, it seems probable that some impairment of frontal lobe functioning is involved; but we cannot rule out the possibility that impairment of behavioral control results from a more generalized interference with cerebral functioning.

A report by Baldwin, Lewis, and Bach (1) is extremely pertinent to any discussion of LSD localization. They found, in a study of chimpanzees, that animals with bifrontal lobectomy have the typical (for chimpanzees) reaction to lysergic acid; after ablation of both lateral temporal cortices, the reaction is either altered or absent. There are obvious problems in interpolating from subhuman to human function, however. For example, about 25 times the human per kg. dose is needed to produce the animal reaction. It may well be that the intellectual and motivational functions studied in humans are absent, obscured, or simply more difficult to recognize in animal reactions.

SUMMARY

Highly significant differences were found between subjects tested on the Porteus Maze Test in the normal state and those tested under either 75 or 100 mcg. LSD-25. The differences were related to a lack of the ability to carry out well-planned and adaptive behavior under the drug. The marked effect under a relatively light dose was taken to indicate an extreme sensitivity of the function to lysergic acid diethylamide. Similarities between LSD results and those found with lobotomy patients were discussed.

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THE DOCTOR-PATIENT RELATIONSHIP IN A PSYCHOPHARMACOLOGICAL TREATMENT SETTING[1]

LEON D. HANKOFF, M.D.,[2] DAVID M. ENGELHARDT, M.D., NORBERT FREEDMAN, PH.D., DAVID MANN, PH.D. AND REUBEN MARGOLIS

Recent progress in psychopharmacological treatment has taken place in a frame of reference largely detached from that of the interpersonal one in psychiatry. The impressive advances in physiological treatment have encouraged an orientation to the problem in terms of classical pharmacological experimentation. With increasing experience, however, it has become apparent that psychopharmacological treatment contains all of the complexities that any treatment in psychiatry contains. Thus, along with the "pure" pharmacological effects of a drug we have come to realize the role of incidental non-pharmacological factors at work in the treatment. Research with placebos, for example, has very pointedly demonstrated the potency of non-pharmacological factors in a treatment which was essentially pharmacologically conceived.

Understanding the doctor-patient relationship in psychopharmacological treatment is a challenging problem, and it is perhaps natural that the general research interest has been in other areas. In contrast to psychotherapy, the doctor-patient relationship in psychopharmacological treatment is not usually subjected to the conscious scrutiny of the participants nor is it usually assumed that the relationship is crucial to the outcome. In addition, the rapid changes often seen with psychopharmacological treatment would suggest that physiological rather than interpersonal events are the important parameters.

Although little emphasis has been placed on doctor-patient interaction during drug treatment, many research designs have included controls for doctor and observer bias; and there have been some studies measuring this effect of the doctor—his role and attitudes on psychopharmacological treatment. For example, Feldman (3) and Uhlenhuth *et al.* (8) have shown that more enthusiastic or confident physicians appeared to get better results with active psychopharmacological agents. In another study, Atoynatan *et al.* (2) demonstrated greater effectiveness of inhalation therapy when the physician adopted a more permissive role toward the patient.

In this study we are interested in the role of the doctor in psychopharmacological treatment, but more specifically the interaction of this role with that of the patient, that is, the doctor-patient relationship and determining what effect the relationship might have on treatment.

METHOD

Treatment Setting: This is a study of 30 patients treated in the Psychopharmacological Clinic of Kings County Hospital. All patients had clear evidence of a schizophrenic illness dating back more than twelve months prior to clinic intake. They were between 17 and 40 years of age, mean 29.5, and willing to cooperate in a course of drug treatment. The level of manifest psychopathology was high and nine out of the 30

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[2] Department of Psychiatry, State University of New York, Downstate Medical Center, Brooklyn, New York. This project was undertaken by the Psychopharmacological Research Unit of the Downstate Medical Center under the direction of David M. Engelhardt, and was supported in part by grants from the National Institute of Mental Health, U. S. Public Health Service (MY 1983), and Wyeth Laboratories, Radnor, Pennsylvania. The promazine used in this study was supplied as Sparine by Wyeth Laboratories.

patients started treatment immediately from a recent hospitalization.

Treatment consisted of medication given in the context of a brief psychiatric interview. All patients received placebo capsules T.I.D. for three weeks and were then placed on active ataractic medication (reserpine or promazine). Assignment to an active agent was on a random basis. In terms of the present study with its small number of cases, no distinction is made between the reserpine and promazine-treated patients and both will always be considered together. The individual patient was assigned to a particular psychiatrist whom he saw regularly. The initial interview was a fairly extended diagnostic session, while subsequent meetings were much briefer. After three weekly interviews, the interval of appointments was progressively lengthened until a fixed interval of one month was established.

This design, utilizing a three weeks baseline placebo period, has been found most useful to us in the study of incidental treatment factors, such as the doctor-patient relationship, because it allows us to compare subsequent response to an active agent with the earlier placebo response to incidental factors when the specific agent has not yet been administered.

Assessment of the Doctor-Patient Relationship: As our approach to the doctor-patient relationship, separate methods were devised for gauging the attitudes of the respective participants, doctor and patient, at the initial interview.

1. Denial Rating. Immediately following the initial interview, the psychiatrist rated the patient's attitudes toward his illness and treatment along several dimensions.[3] Among the attitude measurements, the rating pertaining to denial or admission of illness was selected for study here, having shown a close association with attitudes of disinterest or eagerness for treatment, respectively (4). The doctors used a four-point rating scale ranging from "1," maximum denial, to "4," maximum admission.

2. Doctor Relationship Index (RI). In order to objectively evaluate the doctor's response to the patient, a method was devised using a content analysis of the doctor's initial interview write-up. This method, which has been described elsewhere (4), was developed from an approach by Kelly and Fiske (7), and is based on the assumption that the doctor's write-up may be used as a reflection of the doctor's responsiveness to the patient. A detached attitude on the part of the doctor is indicated by a write-up emphasizing diagnosis, technical evaluation, and classification of the patient. A responsive or "warm" attitude on the part of the doctor is inferred by his attention to the patient's subjective responses and interpersonal relationships. Each thought unit was coded and a ratio or Relationship Index (RI) derived, with the higher index indicating a "warmer" doctor attitude. The range for the RI was from .14 to .90 for the present group of patients. Based on a previous study, a "warm" doctor attitude was distinguished from a detached doctor attitude by an RI of .60 or above (4).

3. Classification of Relationship. Basic to our study was the conception that the doctor-patient relationship is an emergent event stemming from the doctor's and patient's responses to the interview situation and that both aspects must be considered in defining a relationship. This concept was applied through the use of the dimension of consistency or congruency in classifying the relationship. A consistent relationship was seen as one in which the doctor and patient were similar in their orientation at the initial interview, both being involved and responsive or both being indifferent or detached. An inconsistent relationship was one in which the paired attitudes were contrasting or incongruent. Utilizing the denial rat-

[3] The dimensions were: 1) faith in the treatment program, 2) eagerness to receive treatment, 3) externalizing of illness cause, 4) amount of subjective discomfort, 5) concern about mental health, and 6) acceptance or denial of illness.

TABLE 1
Classification of Doctor-Patient Relationship in Terms of Consistency

Patient's Attitude toward Own Mental Illness	Doctor's Attitude toward Patient in Terms of Doctor Relationship Index	
	"Detached" (RI ≦ .59)	"Warm" (RI ≧ .60)
"Denying" (Rating = 1 or 2)	Consistent	Inconsistent
"Accepting" (Rating = 3 or 4)	Inconsistent	Consistent

ings we classified the patients as to their acceptance (score, 3–4) or indifference (score, 1–2) toward treatment. The RI was used to designate the doctor as "warm" (score, .60 or above) or "detached" (score .59 or below). The combined denial rating and RI provided us with an operational classification of the relationship (Table 1).

Placebo Response: The patient's response to the placebo during the first three weeks of the study was assessed by an independent evaluation of the doctor's progress notes. A placebo response was defined as a change not present before the treatment that was observed by the psychiatrist or verbalized by the patient, and associated by the patient to the use of the pill. By a method described elsewhere (6), we distinguished between favorable or positive response, unfavorable or negative response, and absence of response to placebo. Other aspects of the placebo response which were also rated, such as duration or symptoms affected, are not pertinent to the present study.

Clinic Course: An objective and meaningful criterion for outcome in an ambulatory schizophrenic patient is his ability to maintain himself in treatment. For this purpose, the patient's clinic course, evaluated two years after intake, was used. This included three possibilities: continuation (two years active treatment), nine patients; spontaneous drop-out, 18 patients; and hospitalization for mental illness, three patients. The latter two groups, drop-out and hospitalized, will be considered together as treatment "failures" as their leaving treatment was not medically sanctioned, in contrast to the continuers or treatment "successes."

ANALYSIS OF DATA

The consistency dimension of the doctor-patient relationship had been investigated earlier as a factor affecting the course of a patient in psychopharmacological treatment (4). In the previous study it was found that the type of relationship seen in the initial interview was of high predictive value for the patient's future in treatment: a consistent relationship was associated with continuation; an inconsistent relationship, with spontaneous drop-out from treatment. The placebo response had also shown a meaningful relationship to clinic course when studied in the design described above (6). In a separate study, it had been demonstrated that the absence of a favorable placebo response was strongly indicative of eventual treatment "failure," that is, drop-out or hospitalization.

We are thus presented with two incidental factors, doctor-patient relationship and placebo response, both of known influence on the course of psychopharmacological treatment. Knowing these two factors, we are now concerned with their interaction and what the contribution of each factor is to the final treatment outcome.

As a preliminary step to the examination of the interrelation of the two incidental factors we considered the possible correlations of the components of the doctor-patient relationship, that is, the denial rating and the RI. Neither of these measures revealed any significant relationship with placebo response, clinical course, or psychopathology ratings for the 30 patients.

In investigating the interrelation of doctor-patient relationship and placebo response, our first consideration was the possibility of an association between the forms of placebo response and the classes of doctor-patient relationship. The patients were divided according to whether the doctor-

patient relationship was consistent or inconsistent, and whether the response to placebo was favorable or non-favorable (negative or absent response). The incidence of the possible combinations was tabulated, and, as shown in Table 2, the distribution appears random for all but one combination. The combination of an inconsistent doctor-patient relationship and a favorable placebo response was seen in only two of the 30 patients. It suggests that an inconsistent relationship precludes a favorable placebo response while the consistent relationship yields no particular trend.

The combined effect of a doctor-patient relationship and placebo response on clinic course was then studied. The inconsistent relationship group which had shown a preponderance of non-favorable placebo responses was found to be essentially a treatment "failure" group. Of the 12 patients in this group, nine spontaneously dropped out of treatment. Among these nine drop-outs were eight non-favorable placebo responses.

The consistent relationship group of 18 patients which had been found to be equally divided for favorable and non-favorable placebo responses, was next studied for clinic course. As shown in Table 3 a very definite trend exists, depending on the form of the placebo response. Among the nine non-favorable placebo responses there was uniform "failure," not a single case continuing in treatment, while six out of the nine favorable response patients remained in active treatment.

To summarize our findings, we have distinguished a series of events in the course of psychopharmacological treatment and indicated the interrelation of these occurrences. The first event in the series is the meeting of doctor and patient. Here, our findings confirm a previous conclusion (4) that it is not doctor or patient attitude alone but the combination which is significant for clinic outcome. Consistency of the doctor-patient relationship is seen as a highly favorable condition for continuation in treatment.

TABLE 2

Doctor-Patient Relationship and Placebo Response

Doctor-Patient Relationship Category	Placebo Response	
	Favorable	Non-favorable
Consistent (N = 18)	9	9
Inconsistent (N = 12)	2	10
Total	11	19

TABLE 3

Placebo Response and Clinic Course for Patients with Consistent Relationships

Placebo Response	Clinic Course	
	"Success" (Continuation)	"Failure" (Drop-out, Hospitalization)
Favorable	6	3
Non-favorable	0	9
Total	6	12

$\chi^2 = 6.25$, $p < .02$, using Yates' correction for continuity.

The findings with placebo response suggest an intermediate phase. When the doctor-patient relationship is not consistent, a favorable placebo response is almost completely precluded (10 out of 12 cases). Consistency of relationship, on the other hand, is not predictive, and the type of placebo response is evenly divided in this group. Continuing our analysis to the last phase, clinic outcome, the interaction of doctor-patient relationship and placebo response emerges. For it is now seen that if a patient is presented with the favored consistent relationship but fails to respond favorably to the placebo, he invariably becomes a treatment "failure."

The consistency of doctor-patient relationship seems to act as a limiting factor or *sine qua non* in the psychopharmacological treatment of schizophrenic outpatients. Without consistency in the relationship the patients do not have favorable placebo responses and do not maintain themselves in

treatment. In the case of patients involved in a consistent relationship, the future remains open and depends heavily on the patient's responding favorably to placebo. The question of what factors determine the form of the placebo response in the consistent group remains unsettled. It may be that extra-treatment factors or individual dynamic configurations determined the response to placebo. The favorable and non-favorable placebo responders were compared on base-line psychopathology, but no distinction emerged.

DISCUSSION

The treatment atmosphere of this study was not geared toward an "intense" doctor-patient relationship in the conventional sense. There are several reasons for this. To begin with, the psychiatrist's treatment was conceptually centered around drugs. The meetings with the psychiatrist were brief and often routinized. The patients, for their part, had limited educational backgrounds (the majority had not been graduated from high school), were drawn from the lower socio-economic classes, and had considerable impairment due to their illnesses. Verbalization and introspection were thus limited from both sides of the relationship.

In view of this dampening of a verbal relationship, the findings take on a particular meaning. The schizophrenic outpatient is seen here as responding strongly to a situation which greatly de-emphasizes the verbal, psychotherapeutic aspect of the relationship. The schizophrenic patient's responsiveness to non-verbal cues is here again confirmed. It is our theory that the response set of the patient to the interpersonal climate is the substrate in which the treatment operates. Under the influence of the consistent relationship, a particular set or ego state develops in which the patient becomes available for further clinical change. Or, as in the case of the inconsistent relationship patients, further change is precluded by the lack of such a set. We think of this set or ego state as a stage in which the patient permits an involvement with an external object. This involvement is subtle and limited but quite basic to the patient's defensive structure. It may be likened to the transitional object relation of the infant which precedes the first true object relation (9). Both the infant and the patient relax some of their defensive efforts and try out a relationship in an uncommitted form. There then ensues a reorganization in relation to external reality and to internal defenses. The reorganization of relationships and defenses may then go on to a selection of new defenses with the emergence of the phenomenon of clinical improvement. Psychopharmacological treatment with its oral medication serves as a transitional object in this course of events, a halfway mark toward a meaningful object relation. The temporary lability of relationships and defenses is the response set in which the treatment exerts its specific effects.

Gliedman *et al.* (5), in offering a conditional reflex explanation of placebo response, have suggested that the doctor's ability to relate favorably to the patient may create a "central excitatory state," a base-line state necessary in the production of a conditional reflex. Our concept of the doctor-patient relationship as a required starting point for subsequent treatment response bears some resemblance to their formulation. The theory is here not strictly applicable because our finding concerns a doctor-patient relationship combination not explainable as a particular doctor stimulus quality or effect.

We feel that our findings contain a definite implication for the clinical management of schizophrenic outpatients. Although the relationship findings are in terms of a pairing of attitudes and not based on the attitude of either member taken alone, the psychiatrist is in a position to attempt to alter his role. Using the level of the patient's denial as a barometer, the psychiatrist might vary his own treatment approach to reflect that of the patient. In the development of flexible

and brief psychotherapies, workers have emphasized the need for early dynamic formulation and therapist responsiveness (1). A rapid cementing of the patient's ties to the treatment is particularly important in the psychopharmacological treatment setting where the patient's attachment is so tenuous and treatment failure so frequent. The use of a denial evaluation by the psychiatrist appears to be a valuable rule of thumb for quickly adjusting his orientation to that of the patient. Also of some significance is the usefulness of the placebo response in understanding the initial doctor-patient relationship and its role as a limiting factor in treatment course.

CRITIQUE OF THE STUDY

1. The findings are entirely dependent on the psychiatrist's judgment—his rating of the patient's denial and his interview notes. Furthermore, the psychiatrist, who rates the patient, may considerably affect the patient's level of denial through his manner of interviewing. The study is lacking in objective third-party assessment of doctor and patient. This limitation is somewhat offset by the method of the objective analysis of the notes and by the fact that the psychiatrist had no awareness that his notes would be so used.

2. Our definition of a "warm" or accepting doctor orientation is somewhat incomplete. In applying the measure of the RI to each set of notes, there is the implication that a doctor may show a wide range of relationships. This cannot be definitely concluded from our data. Although the overall range of RI is wide, nine different doctors were included in the study and the number of patients for each doctor does not permit any conclusion as to the individual doctor's range of RI. It is possible that a doctor may have a rather limited range in his relating and present the same orientation to all patients. We are in the process of studying the differential response of doctor to patient.

3. The findings of the influence of the doctor-patient relationship occurred in a design which started treatment with a baseline period of placebo usage. The use of an active psychopharmacological agent at the inception of treatment may vary the influence of the observed factors considerably. Zucker *et al.* (10) have demonstrated (with opiate withdrawal syndrome) that placebo or ineffective drugs may adversely affect the course of the illness. The sensitivity of our patients to the relationship may have been due to an adverse effect of placebo. A comparison of the influences of placebo and active drug on doctor-patient relationship might serve as a good test of a specific psychopharmacologic action of a drug.

4. No rating of clinical improvement has been used in the present study. Our criterion of overall clinic outcome is a crude and limited one. There may have been considerable variation in clinical status within the "success" group. Furthermore, the drop-out group may have contained patients who were much improved. On the other hand, criteria of clinical improvement based on psychiatric judgment have often been misleading. We have not solved the dilemma but have used the criterion which appears the most unambiguous in its measurement.

5. The factors of social class and diagnosis are highly significant variables. The patients in this study were drawn from the lower socio-economic classes and had evidence of a schizophrenic illness of over a year's duration. Any generalization in this study must be made in relation to these two factors and this selected patient group. On the other hand, we consider this study as having pointed up the applicability and meaning of our form of treatment for a large segment of the schizophrenic outpatient population.

SUMMARY

The doctor-patient relationship in the initial interview was studied for 30 schizophrenic outpatients receiving combined drug and brief psychotherapy. The doctor and patient aspects of the relationship were

objectively assessed and the paired ratings used for a designation of *consistent* or *inconsistent* treatment relationships.

An *inconsistent* doctor-patient relationship was found to militate against a favorable response to placebo (10 out of 12 cases), as well as against continuation in treatment (nine out of 12 dropped out). When the relationship was *consistent*, the placebo responses were divided between favorable and unfavorable responses, there being nine of each. In terms of clinic outcome, all nine unfavorable placebo responders in the *consistent* group were found to be treatment "failures," either dropping out or being hospitalized. Among the favorable placebo responders, six out of the nine continued in active treatment. We have thus demonstrated a sequence of events and their relatedness in psychopharmacological treatment: initial doctor-patient relationship, placebo response, and ultimate clinical outcome.

We view the schizophrenic outpatient in psychopharmacological treatment as being extremely sensitive to the initial interview relationship and placebo factors. When presented with a *consistent* relationship, if he is unable to respond positively to an oral placebo, active treatment involvement is precluded.

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STATISTICAL CONTROL STUDIES IN NEUROLOGY

X: Relationship between Frequencies of Reflexes in a Group of 2500 Non-Neurologic Patients: Babinski, Hoffmann, Rossolimo, Oppenheim, Gordon and Absent Cutaneous Abdominal Reflexes

M. J. MADONICK, M.D.[1]

The incidence of the Babinski, Hoffmann, Rossolimo, Oppenheim, Gordon and absent cutaneous abdominal reflexes in the same group of patients admitted to a general hospital have been reported individually in previous communications (1–6). The object of the present study is to determine by statistical methods the relationships between these neurological signs.

MATERIALS AND METHODS

The group examined for the above signs was composed of 2,500 patients with non-neurologic disorders, admitted to the surgical, medical and obstetric wards of the Morrisania City Hospital in 1935 and 1936. There were 1695 females and 805 males. No hospital records were used. All examinations were carried out by the present investigator. The wards were visited in rotation so that sufficient time would elapse between return visits to permit discharge of previously examined and the arrival of new patients. The methods of elicitation and reactions that were considered positive were described in detail in the individual paper; there follows a brief summary of the methods employed in testing for the various signs.

The Babinski sign was considered present when there was a definite and constant dorsiflexion of the large toe on stimulation of the outer part of the sole of the foot with a safety pin; stimulation was adequate but not nocuous. The Hoffmann sign was elicited by snapping the nail of the partially extended middle finger; flexion of the thumb was required for the response to be considered positive. The Rossolimo sign was considered present when there was plantar flexion of the small toes upon tapping of the digital tendons with a heavy reflex hammer on the sole near the metacarpophalangeal joints. The Oppenheim sign was present when the large toe dorsiflexed on energetic pressure produced on the inner aspect of the leg along the posterior edge of the tibia by the thumb of the examiner. In testing for the cutaneous abdominal reflexes the dull end of a safety pin was first employed; if no response was demonstrated, then the sharp end of the pin was substituted. The direction of the stroke was from the lateral margin inward and was carried out quickly. The cutaneous abdominal reflex was classified as absent, when there was no contraction of the abdominal wall in the area stimulated. In testing for the Gordon sign, with the patient lying in bed and the feet slightly rotated laterally, deep pressure on the calf muscles was produced by compression between the fingers of the examiner; dorsiflexion of the big toe denoted a positive response.

RESULTS

The frequency of each of the signs with relation to every one of the other signs in turn is shown in 2 × 2 contingency tables that are reported in Table 1. From the figures in the margins of the cells, the following is the order, from the least to the most frequent: Gordon, 33 cases (1.3%); Hoffmann, 52 (2.08%); Oppenheim, 55 (2.2%); Rossolimo, 63 (2.5%); Babinski, 107 (4.3%); absent cutaneous abdominal reflexes, 413 (16.5%). The chi-squares that these comparisons yielded, together with the

[1] Neuropsychiatric Service, Morrisania City Hospital, New York.

TABLE 1

Intercomparisons: Six Neurological Signs

		Babinski			Hoffmann			Rossolimo			Oppenheim			Gordon		
		−	+	x^2	−	+	x^2	−	+	x^2	−	+	x^2	−	+	x^2
Hoffmann	+	44	8													
	−	2349	99	18.8 $p < .01$												
Rossolimo	+	61	2		61	2										
	−	2332	105	.2 $p < .10$	2387	50	1.12 $p < .30$									
Oppenheim	+	41	14		52	3		53	2							
	−	2352	93	66.8 $p < .01$	2396	49	5.1 $p < .05$	2384	61	.93 $p < .50$						
Gordon	+	26	7		32	1		32	1		17	16				
	−	2367	100	27.7 $p < .01$	2416	51	1.00 $p > .30$	2405	62	.52 $p > .30$	2428	39	449.5 $p < .01$			
Absent cutaneous abdominal	+	371	42		407	6		406	7		395	18		407	6	
	−	2022	65	41.06 $p < .001$	2041	46	9.8 $p < .01$	2031	56	1.38 $p < .2$	2050	37	10.6 $p < .001$	2060	27	.68 $p > .30$

TABLE 2

Percentage Distribution of Six Reflexes and Total Sample by Age Groups

Reflex	0–9 years	10–19 years	20–29 years	30–39 years	40–49 years	50–59 years	60–69 years	70 plus
Babinski	1.1 (N = 1)	2.2 (N = 8)	1.3 (N = 7)	1.6 (N = 8)	5.7 (N = 22)	8.5 (N = 27)	9.2 (N = 21)	13.5 (N = 13)
Hoffman	1.1 (N = 1)	0.5 (N = 2)	0.6 (N = 3)	2.1 (N = 10)	3.1 (N = 12)	3.5 (N = 11)	1.3 (N = 3)	10.4 (N = 10)
Rossolimo		6.9 (N = 6)	4.3 (N = 16)	2.8 (N = 15)	2.8 (N = 11)	2.2 (N = 7)	2.6 (N = 6)	2.1 (N = 2)
Oppenheim	2.2 (N = 2)	0.3 (N = 1)	1.3 (N = 7)	1.2 (N = 6)	2.6 (N = 10)	3.5 (N = 11)	6.1 (N = 14)	4.1 (N = 4)
Gordon	1.1 (N = 1)	1.1 (N = 1)	0.6 (N = 3)	0.4 (N = 2)	1.6 (N = 6)	1.9 (N = 6)	1.9 (N = 11)	3.1 (N = 3)
Absent cutaneous abdominal		2.7 (N = 10)	8.4 (N = 45)	13.9 (N = 67)	17.8 (N = 69)	25.8 (N = 82)	36.7 (N = 84)	58.3 (N = 56)
Total	3.5 (N = 87)	14.6 (N = 368)	21.3 (N = 533)	19.2 (N = 482)	15.5 (N = 387)	12.7 (N = 318)	9.1 (N = 229)	3.8 (N = 96)

corresponding significance levels, are also presented in the table. No distinction is made between signs present on the right side, or left side, or both sides or on either side.

Table 2 presents the percentage distribution of each of the six signs, according to age groups. (Observed frequencies—number of patients with the signs—are reported in brackets.) Table 2 also shows the percentage distribution by age groups of the entire sample of 2,500 patients examined (bottom line).

The chi-square comparisons yielded significant relationships as follows: between the Babinski and absent cutaneous abdominal reflexes; between the Hoffmann and

TABLE 3

Relationship of Signs With Age

Sign	χ^2	df	p
Babinski	81.9	7	$<.001$
Hoffmann	54	7	$<.001$
Rossolimo	12.8	7	$<.10$
Oppenheim	29.908	7	$<.001$
Gordon	41.4	7	$<.001$
Absent cutaneous abdominal	73.8	7	$<.001$

absent cutaneous abdominal reflexes; between the Oppenheim and absent cutaneous abdominal reflexes; between the Babinski and Hoffmann signs; between the Babinski and Gordon signs; between the Babinski and Oppenheim signs; between the Oppenheim and Gordon signs; and perhaps between the Oppenheim and Hoffmann signs. No significance was demonstrated between the frequencies of the Babinski and Rossolimo signs, between the Oppenheim and Rossolimo signs, between the Rossolimo and Hoffmann signs, between the Rossolimo and absent, cutaneous abdominal reflexes and between the Rossolimo and Gordon signs. The absence of a significant relationship between the above-mentioned signs does not, of course, necessarily rule out clinical value of these signs. Chi-square tests were also performed to determine whether there is a variation in the probability of the occurrence of each sign as a function of age. With the exception of the Rossolimo, such a variation was demonstrated with each of the other signs, as summarized in Table 3. It was somewhat surprising to find the lack of significance between the frequency of the Rossolimo sign and the frequencies of the other reflexes studied. This result was unexpected since the Rossolimo, as the other reflexes, is considered a sign of cortico-spinal tract involvement. A relationship might be observed between the Rossolimo and the other reflexes if another population, patients with cortico-spinal disease, for example, was examined. Such a study would certainly be of interest.

SUMMARY

The chi-square test procedure was employed to determine whether there is a statistical association between the frequencies of the Babinski, Hoffmann, Rossolimo, Oppenheim, Gordon and absent cutaneous abdominal reflexes in the same 2,500 non-neurologic patients; these signs were reported individually previouslv.

Statistical significance was obtained between the frequencies of the Babinski and absent cutaneous abdominal reflexes, between the Hoffmann and absent cutaneous abdominal reflexes, between the Oppenheim and absent cutaneous abdominal reflexes, between the Babinski and Hoffmann signs, between the Babinski and Gordon signs, between the Babinski and Oppenheim signs, between the Oppenheim and Gordon signs and perhaps between the Oppenheim and Hoffmann signs; no significance was established between the frequencies of the Babinski and Rossolimo signs, between the Oppenheim and Rossolimo signs, between the Rossolimo and Hoffmann signs, between the Rossolimo and absent cutaneous abdominal reflexes and between the Rossolimo and Gordon signs.

Except for the Rossolimo sign, there was a variation in the probability of the occurrence of each of the other signs as a function of age.

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PROLONGED EFFECT OF CHLORPROMAZINE: EEG FINDINGS IN A SENILE GROUP

J. M. SWAIN, M.D.[1] AND E. B. LITTERAL, M.D.[1]

The purpose of this report is to present the evidence of chronic or prolonged effects of chlorpromazine administration, as indicated by sustained EEG changes in a group of elderly women included in a double blind study in the Agnews State Hospital a few years ago. The primary intent of the study was to evaluate the effectiveness of chlorpromazine in altering the behavior of a hospitalized geriatric population. Because there have been relatively little data published on the chronic effects of the phenothiazine drugs, the EEG findings which were noted during the course of the study would seem to warrant some comment.

METHOD

The entire study group consisted of 61 patients of age 65 or over, 31 of whom received chlorpromazine and 30 an inactive preparation of identical appearance, supplied by the same drug firm. The study was conducted in the usual double blind method. Diagnoses were either cerebral arteriosclerosis or senile dementia. An effort was made to eliminate those patients whose personality aberration seemed principally of non-organic origin, such as those with depressive reaction, or paranoid ideation without intellectual deficit. Electroencephalograms were obtained preceding administration of medication and subsequently at approximately one, three and six month intervals. The EEG records were read blind by one of the writers, who had no other association with the project.

RESULTS

Tabulation of the EEG reports at completion of the study yielded the following summary.

Chlorpromazine group:

1. Amplitude increased in 81 per cent, was unchanged in 19 per cent.
2. Predominant frequency decreased in 45 per cent, remained the same in 55 per cent.
3. Paroxysmal characteristics increased in 23 per cent, remained unchanged in 77 per cent.
4. A generalized dysrhythmia initially present increased in 61 per cent, decreased in 10 per cent and remained unchanged in 29 per cent.
5. Of four patients showing focal characteristics at the onset, three showed an increase in focal characteristics and one remained the same.
6. Nineteen per cent of the patients in this group (six of 31) exhibited persistence of the EEG changes, which had developed during the course of the study, for a period of three months or longer after the drug was terminated.

Control group:

1. Amplitude increased in 27 per cent, decreased in three per cent, remained the same in 70 per cent.
2. Predominant or basic frequency remained the same in 94 per cent, decreased in six per cent.
3. Paroxysmal characteristics increased in three per cent, decreased in three per cent, and remained unchanged in 94 per cent.
4. Generalized dysrhythmia characteristics increased in 10 per cent, decreased in six per cent, and remained the same in 84 per cent.
5. Of five patients showing focal characteristics at the onset, two showed

[1] Agnews State Hospital, San Jose, California.

exaggeration in this finding during the study, and one remained the same.

The following case reports are demonstrative of the EEG changes noted with chlorpromazine which persisted for three months or longer after termination of the drug, and which we feel are attributable to the drug effect.

CASE REPORTS

Case #1: This patient was admitted in February, 1951, at the age of 73 (and was 78 at the onset of this study). She presented a ten-year history of memory loss, ideas of reference, restlessness, and possible auditory hallucinations. The patient had been under treatment for hypertension for 10 years. B.P. was 168/94, with rapid auricular fibrillation on initial exam. Mild retinal arteriosclerotic changes were noted. She was digitalized and treated for an episode of pneumonitis in the period preceding this study. The pre-treatment EEG: normal amplitude, containing mixed fast and slow frequencies without organized background rhythm—generalized dysrhythmia. The patient was started on chlorpromazine 10 mgm. t.i.d., with the dosage gradually increased to a maximum of 100 mgm. b.i.d. on 8-29-56, and terminated on 9-12-56. EEG was recorded on 9-5-56 and showed a marked change with amplitude doubled or more, and very marked increase in slow frequencies. Observed were many high-amplitude 2½ cycle waves which at times had a paroxysmal occurrence. An EEG dated 11-1-56 again showed much more slowing than the pre-treatment recording, with continued amplitude increase and slow spikes noted. An EEG on 1-29-57, more than four months after termination of the chlorpromazine, showed a return to a pattern similar to that of the pre-treatment record but with persisting increase in amplitude. It is of interest that at this time the patient's general condition had worsened, and the patient was described as seriously ill with symptoms of gastroenteritis. Despite this the EEG appearance was changing in the direction of lessened abnormality, suggesting that other factors in the patient's condition were not primarily effecting the EEG changes observed. (This patient is still living.)

Case #2: This patient was admitted in June, 1955, at the age of 88, and was described as excited, restless, combative, noisy, and subject to frequent falls. A history of a "heart spell," several years previously was given. Pre-treatment EEG: a rambling, disorganized asymmetrical record with flattening and slower rhythms from the left temporal area—generalized and focal slowing. The patient was started on chlorpromazine in small doses on 7-17-56 and gradually increased to a maximum dosage of 100 mgm. t.i.d. on 8-13-56, with this treatment being discontinued on 10-3-56. A EEG obtained on 8-20-56 showed a severe degree of slowing

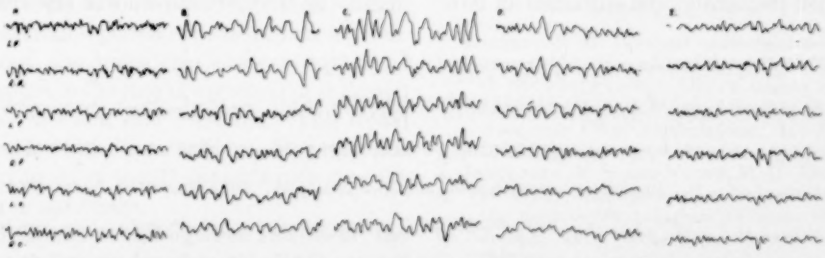

FIG. 1. Case #1. Age 78. A: Pre-treatment recording. B: One month after start of medication (150 mgm. daily at time of EEG). C: Three months on chlopromazine (25 mgm. daily at time of recording). D: Twelve weeks after termination of drug, showing persisting amplitude increase and slowing. E: Twenty-five weeks after termination. (All recordings with comparable standardization, illustrations selected from monopolar runs with ear on same side as reference point. Temporal leads omitted from tracings).

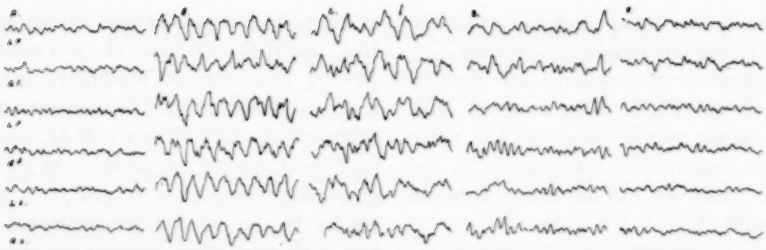

FIG. 2. Case #2. Age 90. A: Pre-treatment record. B: One month on chlorpromazine (300 mgm. daily at this date). Patient awake. C: Three months on drug (10 mgm. daily—reduced from 300 mgm. daily seven days preceding this recording). D: Twelve weeks after termination of the drug. E: Twenty-four weeks after termination of the drug. Note that high amplitude dysrhythmic characteristics have subsided.

and an increase of amplitude. The EEG taken on 10-10-56, three months after the start of medication and one week after termination, showed continued marked increase in abnormality, with amplitude greatly increased, 2–3 cycle high amplitude irregular forms. An EEG dated 1-21-57, more than three months after termination of the drug, was reported to resemble the pre-treatment recording, despite the fact that the patient had declined in general condition and was at this time partially bedridden. (This patient subsequently died.)

Case #3: This patient was admitted in April, 1954, at the age of 80 (and was 82 when included in the study). She had a 20-year history of hypertension and was forgetful, resistive and restless. The physical examination on admission showed a slight right-sided spasticity and a positive right Babinski sign. The patient was unsteady in gait, fell frequently, and sustained an intertrochanteric hip fracture in May, 1956, with fairly good recovery following an open reduction. Pre-treatment EEG: good amplitude, fairly well-organized record with predominant frequency at 7 to 7½ cycles per second—generalized slowing. She was started on chlorpromazine 10 mgm. t.i.d. on 6-12-56, increased to maximum of 100 mgm. t.i.d. on 7-10-56, and then gradually reduced, with the last dose given on 9-3-56. An EEG obtained on 7-10-56 showed a generalized paroxysmal slowing with focal slowing over the left mid-hemisphere and a well-organized rhythm on the right (interpreted as chlorpromazine activation of focus compatible with evidence of pyramidal tract involvement that had been noted on admission examination two years previously). EEG recordings on 8-8-56 and 9-5-56 were similar, with marked abnormality continuing. An EEG on 2-18-57, five months after termination of chlorpromazine, was reported as

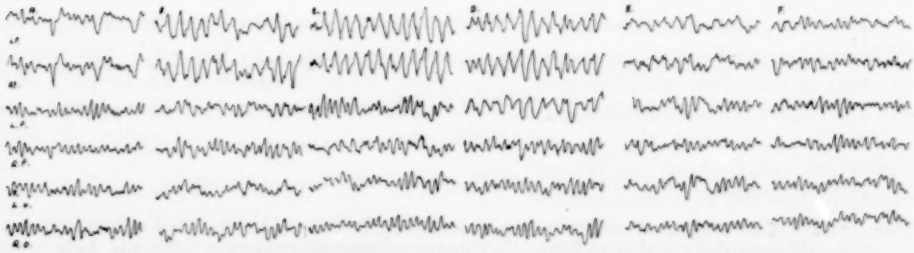

FIG. 3. Case #3. A: Pre-treatment record. B: One month after start of chlorpromazine (300 mgm. daily). C: Three months on drug (150 mgm. daily). D: Four months on drug (25 mgm. daily). E: Twelve weeks after termination of drug. F: Twenty weeks after termination of drug. Record returning to pre-treatment pattern.

improved in direction of return to pre-treatment pattern, but still showed more slowing than initial record. (This patient is still living and her general condition is much the same as described on admission.)

DISCUSSION

That chlorpromazine can produce EEG abnormalities or accentuate previously existing abnormality is known (2, 3) and in fact parenteral administration of chlorpromazine to highlight focal abnormalities has been used in some EEG laboratories. Promazine has a similar effect, while some others of the phenothiazine group seldom produce EEG changes. The frequent occurrence of slowing of the basic frequency and increase in voltage agrees with findings mentioned by Szatmari (5). It is also known that elderly patients are often particularly sensitive to the effects of the drug, and, as seems to be true in all age groups, there is considerable individual variation in the susceptibility or tolerance to chlorpromazine and other phenothiazines. That there may be a cumulative action of the drug with persisting effect for several weeks after the ingestion of the drug has been discontinued, has been observed, perhaps most prominently in the extra-pyramidal syndrome which occurs as a side effect.

Forrest (1) has reported recovery of urinary metabolites of phenothiazine drugs for many weeks after termination of administration and in some patients studied by him, the excretion period extended to six months. Forrest also notes that there is individual variation in the rate of excretion of the drug, with "fast and slow excretors." This would coincide with our findings of prolonged effect in six of the 31 patients receiving the drug in our study.

In the report of Jorgensen and Wulff, the abnormality produced in the EEG records of their cases on chlorpromazine had disappeared at the end of 10 days. This was a group on non-organically involved patients in an age range of 17 to 59 years. Robinson (4) reports deterioration of functioning level in 34.7 per cent of geriatric patients on chlorpromazine, contrasted with 5.3 per cent in patients on placebo, but apparently did not include EEG tracings among other observations in his study. The degree of EEG change noted in our patients while on chlorpromazine may well be related to the age level and presence of brain damage. However, the persistence of the changes for three months and longer in some, but not all of the patients receiving the drug, seems unrelated to the age factor.

CONCLUSIONS

EEG recordings obtained during a double blind study of the effects of chlorpromazine in an aged, brain-damaged group of patients, showed markedly greater incidence of increase in abnormality in EEG recordings in the patients receiving chlorpromazine in comparison with a control group. Increased voltage and decrease in basic frequency were most often observed.

In some instances the changes in the EEG attributed to chlorpromazine persisted for three months or longer after the drug was discontinued. No other factors to account for the persisting abnormalities were identified in clinical evaluation of these patients which would differentiate them from others in the treated or control groups. This suggests cumulative action and delay in metabolism and excretion of the drug. As a corollary to this, the need for sustained release type of dosage form may be questioned.

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BOOK REVIEWS

ARIETI, S., ED. *American Handbook of Psychiatry.* Two volumes. Basic Books, New York, 1959. x + 999 pp. (Vol. 1); ix + 1098 pp., illus., (Vol. 2) $25.00

Many American psychiatrists surely have dreamed of producing a *Handbuch* which would bring order into the chaotic field, and, incidentally, establish themselves for professional posterity. However, psychiatric matters do not easily lend themselves to this form of treatment, so, in spite of a number of lesser attempts at systematization, ranging from pocket-sized synopses, through textbooks such as Noyes', to Fenichel's compendium of psychoanalytic theories of mental illness, the opportunity has not been seized until the present moment. And even here the presentation does not convey the Teutonic sense of finality and rightness that might be expected. Rather, in the American tradition of debate and uncertainty, the tenets of every leading school of thought and major approach are offered to the reader who must, himself, pick and choose. It is, perhaps for this reason that the Handbook fills two thick volumes, occupies over 2000 pages including indices, and is too heavy to carry with comfort.

Silvano Arieti, the editor, has surrounded himself with a distinguished editorial board (Appel, N. Cameron, Balin, K. Goldstein, L. Kolb) and has drawn upon the talents of 111 authors representing varied schools of thought. The effort at broadness and impartiality is impressive; yet, a work of this kind must to some degree promulgate the point of view of its organizer. In this instance there is a heavy representation of authors from the New York City area, particularly those associated with the William Alanson White Institute (of which the Editor is a faculty member), and other groups, such as the American Institute of Psychoanalysis, rightly or wrongly associated with the "interpersonal" or "socio-cultural" in contrast to the classical psychoanalytic points of view. Arieti, himself, contributes four of the six chapters concerned with "The Functional Psychoses."

But this may be carping. Norman Cameron's chapter on "Paranoid Conditions Paranoia" is a joy to read. The section on "Psychosomatic Medicine" with chapters by Lidz, Reiser, Wittkower and others is a strong one. Cramer, Mahler, Adelaide Johnson, Gardner and others write well in the section on "Childhood and Adolescence." Ruesch ("General Theory of Communication in Psychiatry") and Szasz ("Language and Pain") offer typically lucid and thoughtful chapters on the section on "Language, Speech, Communications." Weinstein and Kahn's chapter on "Symbolic Reorganization in Brain Injuries" in this section is an excellent condensation of work upon which they have reported in detail elsewhere. Volume I, in addition, includes sections of several chapters each entitled, "General" (history, personality theory, families, statistics, genetics, interviewing), "The Psychoneuroses and Allied Conditions," and "Psychopathic Conditions, Deviations, Addictions."

Volume II, after 286 pages devoted to brief chapters on "Organic Conditions," is concerned mainly with therapy and picking up the loose ends ("Relations with Basic Sciences and Experimental Psychiatry," "Contributions from Related Fields," "Management and Care of the Patient," "Legal, Administrative, Didactical and Preventive Psychiatry.") The sections on "The Psychotherapies" and "Psychoanalytic Therapies," including five chapters each, will be received with mixed feeling and varying degrees of interest, depending upon the orientation, clinical experience, and sophistication of the reader. Greenson represents "The Classic Psychoanalytic Approach" and the other chapters are concerned with group

therapy, psychodrama, existentialism, psychobiology, K. Goldstein's "Organismic Approach," Horney, Adler, Jung, Rank, "The Interpersonal and Cultural Approaches" and hypnotherapy. The standard physical therapies are discussed in another section. Such solid scientists as Papez, Ralph Gerard and Stanley Cobb attest to the possible relationship of psychiatry to science. Nursing, social work, and family care receive their due, as do various aspects of forensic psychiatry. Even psychiatric teaching of medical students (Maurice Levine) and residents (E. Gildea) is not ignored, and the Handbook closes with chapters on mental hygiene by Lemkau and "The Organization of Psychiatry in the United States" by Blain.

These volumes are presumably intended as reference works. It is quite possible that some residents and students will try to plow through them in an effort to swallow psychiatry at one gulp. They will presumably suffer from indigestion. Senior psychiatrists who are writing papers and preparing lectures will find useful, but not all-inclusive, bibliographies and accounts of their subjects. These accounts should be treated as beginnings and not ends. Some will pick up the volumes at odd moments and browse in areas of their particular interest. Believe it or not, they even contain good bed-time reading, although their size and weight make them awkward. Of general interest are the chapters on "Religion" (Appel, Higgins, Ostow, von Domarus) and on "The Role of Immediate Experience for Dynamic Psychiatry." This last includes subsections on "The sense of immediacy in Fromm's conceptions," "Zen Buddhism" and "Martin Buber."

In summary, one can only repeat that this is the first American Handbook; that, as such, it makes a contribution; and that whether or not there is a need, there will undoubtedly be others. The fact of its existence makes it a necessary acquisition for all psychiatric libraries. Some Departments of Psychiatry are reported to have recommended it for students. This reviewer is not yet certain that students and residents should be urged to make the required financial investment.

Eugene B. Brody

DUNHAM, H. WARREN AND WEINBERG, S. KIRSON. *The Culture of the State Mental Hospital*. Wayne State University Press, Detroit, 1960. xxiii + 284 pp. $5.00

The authors of this book, both sociologists, spent nine months at Columbus State Hospital, Ohio, in 1946, for the purpose of studying how the social organization of a state hospital affects the clinical progress of its patients. In collecting their data, Doctors Dunham and Weinberg relied principally on anecdotal material, gleaned from observations and unstructured interviews with patients and staff members.

Few will argue with the authors' conclusions that the average state mental hospital is primarily a custodial institution; that psychiatric aides act more to restrain than to understand patients; that there exists a fairly rigid social hierarchy in both employee and patient groups; and that patients are often exploited by the hospital and its employees. Having confirmed the obvious, the authors search no further and conclude that the length of hospital stay of many chronic psychotic patients may be seriously influenced by the suppressive atmosphere of the institution.

In attempting to analyze some of the shortcomings of this project, one is tempted to compare it with the Stanton and Schwartz study of Chestnut Lodge (*The Mental Hospital: A Study of Institutional Participation in Psychiatric Illness and Treatment*. Basic Books, New York, 1959). Several important differences are clear. Partly because of the relatively short duration of their study, Dunham and Weinberg were unable to break through the prejudices and suspicions of personnel and employees to the extent achieved by Stanton and Schwartz. As a result, this reviewer's feeling is that the

authors' description of their role as "participant observers" is only half true. Another important difference between the two studies appears to be the relative lack of psychiatric sophistication shown by Dunham and Weinberg. I do not know how much prior acquaintance they had with state hospitals, but they do not seem to have outgrown the initial reaction of most newcomers who identify with the "persecuted" patients and see the staff members as obstacles to their recovery. As a result, one does not get the same objective, sympathetic consideration of the problems and difficulties of the staff which was one of the most valuable fruits of the Stanton and Schwartz study.

The extent of the authors' naïveté in psychiatric matters can best be illustrated by the following quotations. "The psychiatrist, thus, sees an individual who has become mentally ill due to the development of some internal organic pathology; he cannot see a person who, because of an acquired personality organization, cannot function completely and efficiently within his social group." It is difficult to understand how anyone even remotely in contact with current American psychiatric thinking, could make such a statement. One's puzzlement is increased when the authors later state that one-third of the doctors on the staff of Columbus State Hospital "have adapted a psychological dynamic view-point toward the development of functional disorders" (p. 8).

It is equally incredible that the authors found it necessary to make the following clinical observation: "Although psychotics frequently have had many bizarre sex experiences, their breakdowns do not necessarily result from excessive sex activity. In addition, patients such as schizophrenics are very anxious and sometimes even fearful about discussing sexual matters . . ." (pp. 84–85).

In spite of its many weaknesses this book does have a number of virtues. The authors are at least partially successful in capturing the atmosphere of a state mental hospital, and the liberal use of direct quotations from staff members and patients offers an interesting and highly readable departure from the stereotyped descriptive approach.

In assuming that more understanding and sympathetic treatment of psychotic patients will shorten their hospital stay, Doctors Dunham and Weinberg are seemingly stating the obvious. However, as director of a University Psychiatric In-Patient Service, with a doctor-patient ratio of 1:5, I have on some occasions seen our therapeutic failures reintegrate rapidly at nearby state hospitals. Things are certainly not as simple as Doctors Dunham and Weinberg suppose. It is interesting, in this connection, to quote from the conclusions of a study recently reported by an interdisciplinary team of which Doctor Dunham was a member (Amer. J. Psychiat., **116:** 993–1000, 1960): " . . . modifications of hospital culture have little or no impact on the basic schizophrenic psychopathology of chronically disturbed patients."

Walter Weintraub

ROKEACH, MILTON. *The Open and Closed Mind.* Basic Books, New York, 1960. xv + 447 pp. $7.50

This book is the result of a number of individual studies on the social and psychological determinants of belief, studies conducted by the principal author and his numerous collaborators both among colleagues and students at Michigan State University and at other institutions here and abroad. It is an understatement of the good qualities of the volume to say that it compares most favorably in viewpoint, subject matter and method with *The Authoritarian Personality.*

One of the principal merits of *The Open and Closed Mind* is that it goes beyond *The Authoritarian Personality.* While it still is open to many of the criticisms that have been made of the earlier book, it corrects some of the more fundamental mistakes. Rokeach's book begins the difficult and perhaps almost superhuman task of divorcing

studies of this kind from the political, moral and philosophical belief systems of the individuals responsible for the investigations. Of the many weaknesses of *The Authoritarian Personality* perhaps the most obvious was the ease with which the "good guys," those low in authoritarianism, rigidity and their various correlates, could be identified; and it was painfully obvious what the authors of that justly famous study thought of the bad guys.

The investigators responsible for *The Open and Closed Mind* make an effort to deal with the structure of attitudes rather than their content. They deal, thus, with the authoritarianism of the left as well as that of the right. Early in the book the author presents the reader with an interesting essay on the nature of belief (an essay, since it is not in any formal way a theory, nor does the empirical work of the latter chapters appear to depend upon it), and shortly afterwards the stage is set "for studying individual differences in belief systems by describing how and why such systems may vary in the degree to which they are open and closed." It is here that the reader becomes impressed with the difficulty of the task undertaken by these investigators, for it becomes immediately apparent in the parallel descriptions of the open *vs.* closed systems of belief that the authors of this chapter (Rokeach and Restle) have no doubt where their own sympathies are and easily assume that the readers have the same sentiments. After all, no one wants to be accused of having a closed mind, at least no one among the likely readers of this book.

From the account of open and closed systems two empirical, objective measuring instruments are developed. These are the Dogmatism scale and the Opinionation scale. These scales provide the central core of the empirical data presented in this book. They appear to behave very satisfactorily in populations regarded by the authors as being predominantly open or closed minded (of both political wings). Among other things, the authors of the various studies reported later in the book are able to demonstrate, by means of the Dogmatism and Opinionation scales, that the California scales are indeed measures of rightist rather than general authoritarian convictions.

An extended section of this book is devoted to what the authors describe rather grandly as "experimental cosmology." This section, in fact, is devoted to a discussion of how individuals described as being open or closed behave with problems that require them to deal with familiar situations in a novel way. The problems—called a little too coyly the "Joe Doodlebug" problems—are mildly inherently interesting (and thus recommend themselves as intrinsically motivating for subjects), but they are not suitable for analytic treatment. They are, as a matter of fact, so cumbersome that one is puzzled as to why they were not abandoned in favor of some more straightforward way of attacking the same question. One advantage the authors claim for the Doodlebug problems is that they help isolate and separate the ability to break up single closed beliefs (hampered, say, by rigidity) and the ability to break up belief systems (hampered by true closed-mindedness).

Some of the empirical evidence presented in the last third of the book suffers from the defects found in *The Authoritarian Personality*. Indeed, some of the correlations reported here (correlations, for example, between dogmatism and musical taste) make one suspect that the authors have happened on an excellent objective device for separating people they like from people they don't like. More seriously, this points up the general weakness of inferences made from correlational studies. The authors prefer to attribute their correlations to the inherent separation made by the Dogmatism scale; it is possible, however, that the discriminations made by the scale in such things as tastes for Schönberg and Bartok may be determined by conditions not tapped by Rokeach and his collaborators.

More impressive is the attempt to demonstrate the organization of *disbelief* systems (by the skillful use of data on religious affiliation). While it is impossible to summarize this work briefly, it is extremely important. It not only makes a substantative contribution in its own right, but a very considerable important point about the theory and evaluation of attitudes as a whole.

In short, this is an important and useful book. At the same time it shows how far we need to go before we can be said to be genuinely objective with respect to beliefs and attitudes. This book is a great advance over *The Authoritarian Personality*, and perhaps a scientist should ask for nothing more than a steady improvement in technique, theory and objectivity.

James Deese

FROMM, ERICH, SUZUKI, D. T. AND DE MARTINO, RICHARD. *Zen Buddhism and Psychoanalysis*. Harper and Brothers, New York, 1960. 180 pp. $4.00

This book consists of three essays, one by each of the authors. Those by Suzuki and De Martino are almost literal versions of papers presented at a conference in Cuernavaca, Mexico, in 1957. Fromm's paper, according to the Foreword, represents a complete revision of the material presented by him at that meeting.

Suzuki is, of course, the leading spokesman for Zen in the West, and its most prolific exponent in the English language. Those who have read his previous work will find little new in the present essay. For the uninitiated it is a brief introduction to the subject, with a thought-provoking first section delineating the differences between Eastern and Western thought. It begins with a comparison between a *haiku* of Basho, a seventeenth century Japanese poet, concerning an inconspicuous plant blooming along a road, and Tennyson's well known verse about the flower which he plucks. The latter ends:

"Little flower—but if I could understand
What you are, root and all, and all in all,
I should know what God and man is."

Basho does not pluck the flower; he just looks at it. He has no desire to conceptualize it. Tennyson is active and analytical. He plucks the flower from the place where it grows—the plant must die; its destiny is sacrificed to his curiosity. Basho accepts nature: Tennyson resists. His ". . . individuality stands away from the flower, from 'God and man.' He does not identify himself with either God or nature." In contrast to "objectivity," Basho stands for "absolute subjectivity," which excludes empathy, sympathy or identification. This discussion sets the stage for Suzuki's presentation of Zen.

De Martino, the third essayist, is a sometime graduate student in philosophy and religion who has developed his point of view through independent study with a variety of Zen masters including Suzuki. Much of his chapter is concerned with the concept of a "bifurcated ego," which "as affirmer . . . performs the act of affirming itself," while, "as affirmed it is an existential fact presented to itself." The chapter's major appeal for this reviewer lies in its account of the Zen student's progress toward *satori*, or enlightenment, through his efforts to solve a *koan* presented to him by a Zen master. The koan is a problem which offers nothing tangible for the student "to take hold of as object." For example, "What is your original face prior to the birth of father and mother"? The koan "does not permit itself to be fitted into any dualistic subject-object scheme of the ego in ego-consciousness. It can never even be meaningful, much less be 'solved' or satisfied and remain an object external to the ego as subject." The psychoanalytically trained reader will see much in the prolonged painful effort of the Zen student to solve the koan and in his relation to his master during this time, that is reminiscent of the development and ultimate resolution of the transference neurosis. But it implies a kind of absolute involvement that is not encountered

in psychoanalysis: ". . . the authentic struggle with the koan is the struggle of the student to fulfill himself." This struggle is in vain if "undertaken by the ego as subject venturing to deal with . . . its problem as object. For . . . it is exactly the existential dichotomy between subject and object which is the problem of the ego." Ultimately, the koan becomes for the student an expression of the actual "quandary of the ego itself." It becomes, "a living crisis . . . his confronting his own predicament." The master provides for the student "the living assurance of a possible solution . . . the authority, affirmation, and love of the complete fulfillment of the student's very own existence." Finally the ego dies the "great death" which is at once the "great awakening" to "its Self as Self" resolving its initial contradictory duality: "Now it is and knows its 'original face' prior to the birth of its parents . . . this, at last, is Man ultimately realized as Man fully being and having him-Self and his world. . . ."

Perhaps Fromm's essay might have logically followed De Martino, for he attempts a rational comparison of psychoanalysis and Zen Buddhism. Fromm's chapter is the most intellectual of the three, couched in more familiar language, and easier for the psychiatric reader to grasp. He identifies psychoanalysis as "as exquisitely Western as Zen is Eastern," while noting that they both deal with the nature of man and with a practice leading to his transformation. He then goes on to discuss the role of psychoanalysis in "today's spiritual crisis," the values and goals in Freud's concepts, the nature of well-being, and the nature of consciousness and repression. Much of what he says is concerned with differences between feeling and knowing and knowing about, and between thinking of something, and the experience of *being*. Well-being "means to be fully related to man and nature affectively, to overcome separateness and alienation, to arrive at the experience of oneness with all that exists—and yet to experience myself at the same time as the separate entity I am, as the individual . . . to be fully born, to become what one potentially is . . . to be and to experience one's self in the act of being, not in having, preserving, coveting, using."

Freud's system includes concepts basic to the achievement of this state of well-being, which differ from the contemporary Western attitude. Fromm reminds us that Freud wrote of psychoanalytic therapy as consisting in "*the liberation of the human being* from his neurotic symptoms, inhibitions and abnormalities of character," and that the role of analyst transcends that of the doctor who "cures" the patient. "The analyst" Freud wrote, "must be in a superior position in some sense, if he is to serve as a *model* for the patient in certain analytic situations, and in others to act as his *teacher*. . . . Finally, we must not forget that the relationship between analyst and patient is based on a love of truth, that is, on acknowledgment of reality, that it precludes any kind of *sham*, and *deception*." Fromm's italics emphasize the points that he makes with these quotations. He goes on to note three additional aspects of Freud's thought which are closely connected with the Zen point of view: the idea that knowledge leads to transformation, . . . that in the very act of knowing oneself, one transforms oneself; the emphasis on free association versus logical thought; and the willingness to spend years with one patient, implying, in Fromm's words, "that one human being is not commensurable with any thing, that his emancipation . . . is a matter of 'ultimate concern' in itself . . ." Without going into further detail it may be said that Fromm's essay, while it has its carping aspects—particularly when registering his objections to Freud's picture of "man as machine"—will broaden the perspective of the practicing psychotherapist regardless of his particular school of thought.

This is a good book for vacation and bedtime reading. Suzuki and De Martino are tough going much of the way, and the division between philosophy and gobbledygook

is sometimes hard to draw, but the three essays hang together, and they comprise an enormously stimulating and thought-provoking, if occasionally annoying, whole.

Eugene B. Brody

WIRT, ROBERT E. AND SIMON, WERNER. *Differential Treatment and Prognosis in Schizophrenia.* Charles C Thomas, Springfield, Illinois, 1959. xii + 198 pp. $6.50

As the title suggests, the objective of this book is twofold: To present a controlled evaluation of differential treatment in schizophrenia and to study the course and outcome of the disease process itself. Specifically, the authors investigate the comparative effectiveness of chlorpromazine, reserpine, "clinical judgment" therapy (including drugs, electro-shock treatment, insulin and psychotherapy) and routine hospital care in the treatment of eighty previously untreated schizophrenics newly admitted to the Minneapolis Veterans Administration Hospital. Subjects were observed from time of admission to one year following discharge, with overall ratings made at the end of a thirty-day treatment period, at discharge and at follow-up. Since experimental controls were exercised only for the thirty-day treatment period, ratings at the end of this period were regarded as indicative of the relative efficacy of the treatment regimes while those made at discharge and follow-up were viewed as germane to the study of the course of schizophrenia in general. Predictor measures included certain demographic variables, social history factors, an occupational adjustment scale, a behavioral rating schedule, and the Minnesota Multiphasic Personality Inventory. The three latter instruments, together with a global psychiatric rating of improvement, served as criterion measures at subsequent evaluation periods.

With respect to their first objective, the comparative evaluation of treatments, the authors offer a relatively "tight" design, considering that the study was conceived approximately four years ago. Features such as a precisely defined population, the use of acute instead of chronic patients, random assignment to treatment categories, flexible dosage schedules, and an attempt to relate experimental results to follow-up information indicate a decidedly improved approach over much of the treatment evaluation research carried out previously. Even with these features, however, there are a few departures from presently advocated principles of research design which bear mentioning.

The most blatant weakness in the design is the lack of a double-blind technique, which is fully feasible as far as the drug categories are concerned and at least partially feasible in the case of the other categories. As a consequence of this, one cannot accept as uncontaminated the results of the global psychiatric rating of improvement, the major criterion, as a measurement of treatment effectiveness. Rather serious limitations are also found in the statistical analysis of data. Whereas analysis of covariance and more refined mean difference techniques would have been appropriate for much of the data, the authors used less precise procedures throughout. Also, each of the dependent variables was handled separately, with little or no attempt to determine interrelationships among data involving original, subsequent, and change scores. Typical of this approach is the lack of profile analysis of the MMPI except for level comparisons. Evaluation of the results obtained from this instrument are also obfuscated by the discrepant number of subjects on whom successive profiles are based; especially is this true of pretreatment to follow-up comparisons.

Results of the treatment evaluation aspect of this study indicated the relative superiority of the clinical judgment therapy to the others, a finding which holds for all three evaluation periods. Chlorpromazine was found to be an effective drug in the treatment of the schizophrenic, but not as effective as eclectic therapy. Reserpine was found to be no better than routine hospital care.

The authors go so far as to say that reserpine has a detrimental effect when used early in the treatment of schizophrenics, making later treatment efforts more difficult and less successful, and note that for their sample response to the drug was inversely related to both the duration and level of dosage used.

One comment should be made here regarding the relative superiority of the clinical judgment regime. The schizophrenic patients in this study were randomly assigned to the treatment categories regardless of symptoms displayed. From our present knowledge of tranquilizing drugs, we have come to realize the importance of target symptoms in assigning patients to such treatment and would question the meaningfulness of comparing the response of schizophrenics blanketly assigned to tranquilizing drugs to that of schizophrenics assigned to them as specifically prescribed treatment, as in the case of the drug patients in the clinical judgment group. Little is left to chance here since the dice are obviously loaded in favor of tailor-made treatment, whether it be drugs or not.

As for the second objective, the study of schizophrenia in general, the above observations also apply, since there is much overlap in presenting treatment and prognosis material. Inasmuch as experimental controls were not applied throughout the entire observation period, the authors offer some admittedly tentative conclusions regarding the relationship between controlled treatment results and final outcome. These have to do mainly with the beneficial quality of selective treatment and the detrimental nature of reserpine treatment. Most of the authors' findings regarding prognostic indicators are in agreement with past studies. Their results indicate that early treatment of schizophrenics having an acute onset of short duration following a previously stable history of personal, social, and occupational adjustment is most favorable. The chronic nature of the disease is again demonstrated by the finding that outcome at the end of one year following discharge was more often unsatisfactory than not.

Thomas E. Hanlon

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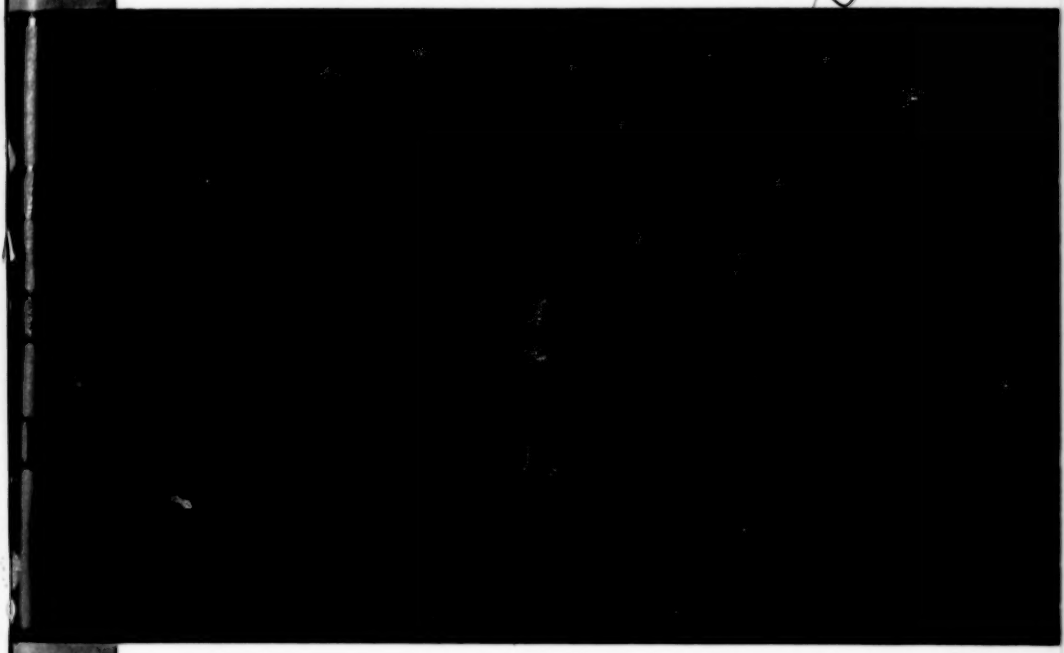

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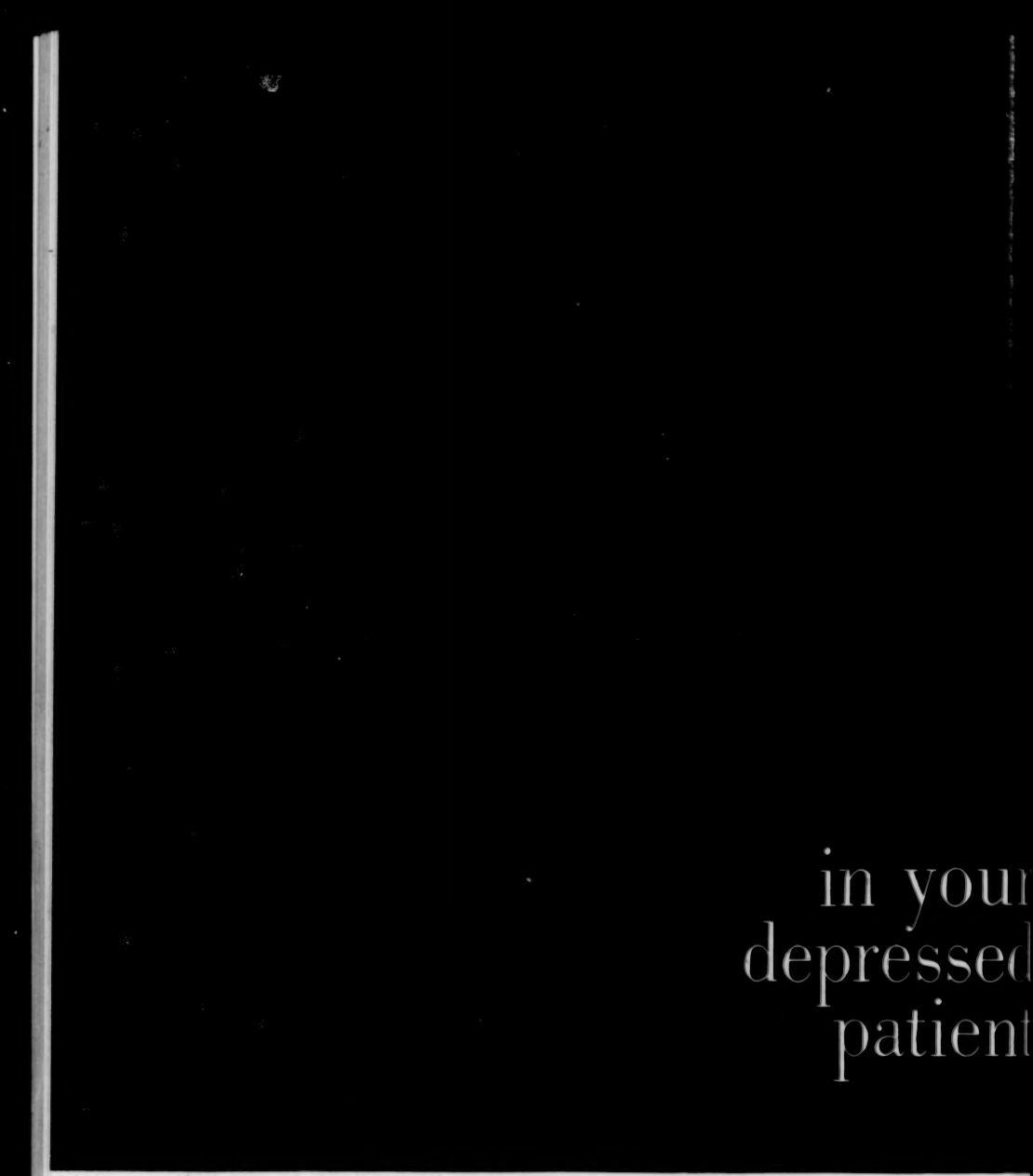

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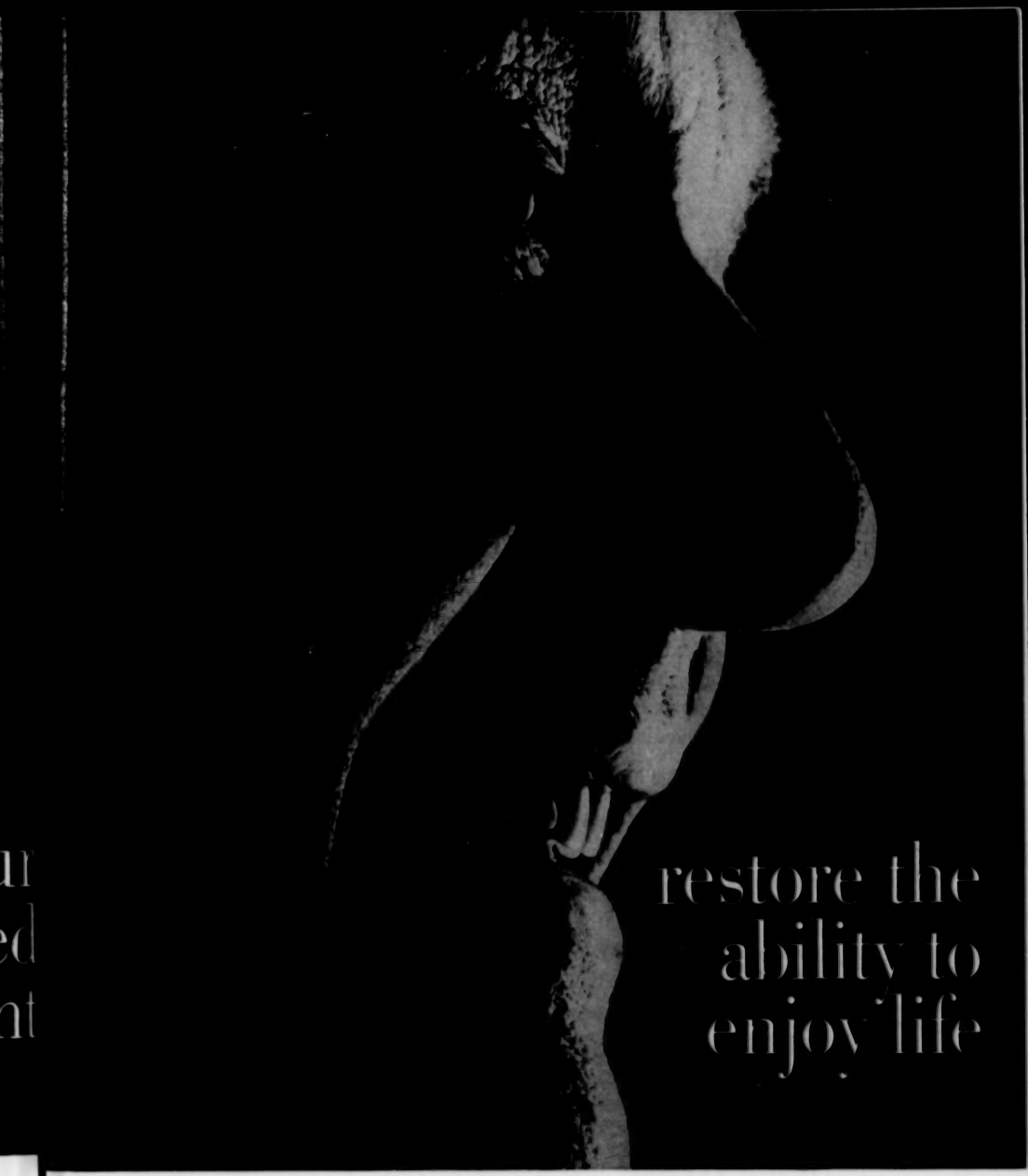

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